Technical Bulletin 105 June 1935

University of Minnesota
Agricultural Experiment Station

A Rickets-Like Disease in Young Cattle

T. W. Gullickson
Division of Dairy Husbandry

L. S. Palmer
Division of Biochemistry

W. L. Boyd
Division of Veterinary Medicine

UNIVERSITY FARM, ST. PAUL

University of Minnesota
Agricultural Experiment Station

A Rickets-Like Disease in Young Cattle

T. W. Gullickson
Division of Dairy Husbandry

L. S. Palmer
Division of Biochemistry

W. L. Boyd
Division of Veterinary Medicine

UNIVERSITY FARM, ST. PAUL

CONTENTS

A RICKETS-LIKE DISEASE IN YOUNG CATTLE[1]

T. W. Gullickson, L. S. Palmer, and W. L. Boyd

Cattle raisers throughout Minnesota suffer severe losses every year from the ravages of a rickets-like disease. The disease usually appears in the winter and spring months, on farms were a liberal feeding of concentrates is practiced. Young cattle, one year old or under, are most frequently affected, but older cattle are not entirely immune. Beef calves seem to be affected more often than dairy calves.

Animals afflicted with the disease exhibit characteristic symptoms. All show more or less stiffness. Usually the forelegs bend forward at the knees and have a tendency to knuckle over at the pasterns. The joints of the leg, especially the hocks, frequently are swollen. Further evidence that the skeleton is affected is the fact that bone fractures are common, especially in the region of the pelvis, and many animals die from this cause. Affected animals usually stand with backs arched and abdomen drawn up. Respiration is invariably rapid, and the affected calf becomes exhausted from even slight exertion. Chronic bloating is of common occurrence and frequently causes the death of an animal. As the disease progresses, all afflicted animals develop anorexia, with consequent loss in weight. Attacks of convulsions are common, and many animals die from this cause.

The disease is not new in Minnesota. Livestock raisers and veterinarians have known of its presence for many years. Similar afflictions in cattle have also been reported in other parts of this country and in foreign lands. In no case, however, has the cause of the disease been definitely established. It was because of this latter fact and because of the unusually large number of affected animals reported in recent years that in the spring of 1930 a study to determine the cause of the disease was begun. The study has been limited to cases occurring naturally in Minnesota and to those produced experimentally at this station. The findings, however, are applicable to similar climatic regions in this and in other countries.

FIELD SURVEYS

Preliminary to the preparation of a plan for an experimental study of the problem, several field trips were made to study conditions on farms were affected cattle were found. About two dozen such farms

[1] The data presented are taken in part from a thesis presented to the faculty of the Graduate School of the University of Minnesota by T. W. Gullickson in partial fulfillment of the requirements for the degree of Doctor of Philosophy.

were visited, and approximately 100 animals afflicted with the disease were observed. Considerable supplementary information was also obtained from local veterinarians and county agricultural agents.

A study of the information obtained from these various sources reveals several facts regarding the conditions under which the disease occurs. It is significant that almost invariably affected animals were fed a ration consisting largely of concentrates with little or no hay. In other cases, the hay was of poor quality and therefore little was consumed. It is noteworthy that even when good quality hay was fed ad libitum, very little of it was consumed, almost all of the animals preferring the more palatable concentrates provided.

This fact strongly suggested calcium as the limiting factor involved, for it is well-known that all concentrates, especially corn, are low in their calcium content, while certain roughages, especially legume hays, are rich in this mineral element. This would also help to explain why the disease is much more common in beef cattle than in dairy animals, because under normal conditions the latter are required to consume considerable quantities of hay. Several farmers reported improvement in affected calves immediately following the addition of alfalfa hay to the ration. A calcium deficiency was further indicated by the fact that affected animals invariably improved after bonemeal was added to the ration.

Further evidence of the probable relationship of calcium to the syndrome is afforded by the following results from the analysis of blood samples obtained from affected cattle at some of the farms visited:

	Blood plasma	
	Ca mgm. per cent	Inorganic P mgm. per cent
Steer; Dawson, Minnesota	10.36	6.24
Steer; Elgin, Minnesota	8.01	8.20
Steer; Elgin, Minnesota	7.77	3.88
Steer; Morris, Minnesota	7.93	2.60

It will be observed that the blood of all except one of these affected animals was below normal in calcium, and several were also low in inorganic phosphorus suggesting the possibility that this element is also a factor. Additional evidence suggesting phosphorus as a factor is the fact that many of the farms represented in the survey are in or are near areas in which the soil is known to be poor in available plant phosphorus. It is illuminating, however, that the symptoms exhibited were not typical of those shown by animals suffering from a phosphorus deficiency.

While these facts suggest the possibility that lack of both calcium and phosphorus may be involved, the situation is further complicated by a possible deficiency of vitamin D or other accessory food factors.

This is apparent because the trouble almost invariably appears during the season of minimum sunshine, or in animals that are kept housed almost constantly. Furthermore, improvement of affected animals is always reported in the spring when sunshine is both more abundant and more effective. In this connection it should be pointed out that the type of ration fed usually changes with the season, and this, as much as the sunshine, might be the effective factor. The fact that increased hay consumption relieved the condition suggests that this part of the ration might also carry essential accessory factors. Furthermore, the improvement reported in animals following bonemeal feeding may not be due entirely to the added minerals, because in the cases reported other changes were usually made in the ration at about the same time.

REVIEW OF LITERATURE

Mineral Deficiency Diseases in Calves

The occurrence of rickets-like diseases in young cattle has been reported by workers in many regions of the world. It is significant, however, that there is little or no unanimity of opinion among these workers as to the cause of such afflictions. Rather, they have attributed the appearance of the disorders to one or more of a variety of causes, depending to some extent, at least, on the nature of the experiment or study in which they may have been interested at the time.

In the United States, Davenport (1897), one of the first to consider nutritive deficiencies in the rations of calves, fed calves on milk alone and on milk and grain. In every case the results were disastrous if hay or roughage of some sort was not included. The symptoms shown by all the animals were about the same: "A ravenous appetite followed by enlargement and stiffening of joints, spells of dizziness and difficult locomotion, all followed by periods of relief and, finally, by a settled feeling of indifference to food. This indifference could be removed temporarily by any change of food, but permanently by coarse food only, which never failed to effect a restoration to natural conditions."

Results of a similar nature have since been obtained by Soxhlet (1901), McCandlish (1918) (1923), Cannon (1931) and others. Reed and Huffman (1924) also found it impossible to raise calves on milk alone, milk and grain, or grain alone. Calves receiving such rations invariably showed symptoms of stiffness and intoxication. In a later report (1926) they state: "In many cases calves fed on concentrates alone develop a condition which resembles rickets in other classes of animals. The legs may become crooked and X-ray photographs of the

ribs may show abnormal calcium and phosphorus depositions." They point out that the cause of this condition is probably a toxaemia, which occurs when a ration composed solely of concentrates is fed.

Olson (1924) carried on tests to determine the relative value of whole milk and skimmilk for calves when supplemented with a free choice of six feeds in self-feeders and alfalfa hay ad libitum. He used five calves. Two of them developed very crooked front legs, and at the end of the experiment one was unable to stand. Inadequate consumption of roughage was given as the cause of all these troubles.

Eckles and Swett (1918) found that a dairy heifer fed a ration low in calcium made normal growth until she was 18 months old, 13 months after being put on the low calcium ration. She then became stiff in the joints and took on an abnormal gait in walking, gradually becoming worse until she walked with her knees partly bent and could get up only with the greatest difficulty. She was restored to normal by the addition of bonemeal to the ration.

Vitamin Deficiency Diseases in Calves

The susceptibility of growing cattle to a vitamin A deficiency was first demonstrated by Jones, Eckles, and Palmer (1926) and later by Bechdel, Honeywell, and Dutcher (1928). These workers state that a deficiency in the vitamin A precursor in the rations of cattle results in xerophthalmia, respiratory troubles and failure to grow. In no case, however, did a post-mortem examination reveal any evidence of bone disease.

Thurston, Palmer, and Eckles (1926, 1929) demonstrated that calves have no apparent need for vitamin C in their rations, and Bechdel, Eckles, and Palmer (1926) found that a calf will grow normally to maturity and produce normal offspring on a ration that carries an insufficient amount of vitamin B to support growth and general well-being in rats. Later Bechdel, Honeywell, Dutcher, and Knutsen (1928) demonstrated that the vitamin B complex or its precursor is synthesized in the rumen of cattle.

On the basis of the rather indifferent effects on calcium metabolism obtained following the administration of various forms of the anti-rachitic factor to mature milking cows, Hart and co-workers (1924, 1926, 1929, 1930) raised the question as to the need for vitamin D. The question might properly be raised, in connection with much of this work, whether the animals used were not already adequately provided with vitamin D in the ration and consequently no definite interpretation should be given to the lack of responses. The same statement might also be used to explain the negative results obtained by Gullick-

son and Eckles (1927), Morrison and Rupel (1927), and Insko and Rupel (1929), with growing calves.

The probable influence of vitamin D, however, was definitely suggested in the study, previously referred to, regarding the vitamin B requirement of the dairy calf, conducted by Bechdel, Eckles, and Palmer (1926).

The need of the growing calf for vitamin D was also indicated by Olson (1926, 1927, 1929). He found that calves fed milk along with free choice of various concentrates showed poor capacity, became stiff, and developed very crooked legs. The stiffness and crooked legs were corrected when calves had access to a greater amount of sunlight. It was found that calves having access to direct sunshine developed heavier bones, weighed more, and were in better physical condition than calves not having access to sunlight. Some of these latter calves became arch-backed, while others developed bowlegs, enlarged joints, and other abnormal conditions.

Huffman (1929) found that adding cod liver oil to a ration of skimmilk, yellow corn, and oats, without hay, permitted calves to grow normally to 28 months of age. Similarly, in studies conducted by Bechdel, Dutcher, and Tucker (1928) and Bechdel and Hill (1930), it was found that exposing either the animal or their rachitogenic ration to the rays of a carbon arc lamp or providing a cod liver oil supplement resulted in a 5 per cent increase in ash deposition.

The reports of Huffman (1932), Rupel, Bohstedt, and Hart (1933), and Bechdel and associates (1933) which appeared during the last few months of the study reported in this bulletin have definitely established the need of young cattle for vitamin D. Huffman found that vitamin D is needed by calves for proper bone-building. He lists the following symptoms resulting from feeding rations deficient in the antirachitic vitamin: 1) anorexia; 2) stiffness; 3) bowlegs—occasionally broken bones; 4) abnormal blood analysis, low blood calcium, or low blood inorganic phosphorus, or low blood calcium and inorganic phosphorus. These symptoms in calves were prevented and cured by: 1) sunshine; 2) ultraviolet irradiation; 3) cod liver oil; 4) viosterol; 5) timothy hay cured in the sun.

Rupel, Bohstedt, and Hart (1933) concluded that calves need vitamin D in some form and incur "rickets" when this vitamin is lacking. They report that calves fed rations deficient in vitamin D, but adequate in other respects, gradually become stiff and slow in their movements, standing with their knees bent forward and displaying an unwillingness to arise when lying or to move when standing. The long bones of the legs become more or less deformed. Others show twist-

ing of the pasterns and enlargement and stiffness of the joints. In several instances convulsions occurred in the more emaciated animals. The blood serum of affected animals showed that both the calcium and inorganic phosphorus were below normal.

Bechdel and coworkers (1933), on the basis of work covering a period of five years or more, concluded that the antirachitic vitamin is essential to the growth and well-being of calves. They found that calves fed a ration which consisted of whole milk from birth to 30 days followed by skimmilk and a grain mixture made up of equal parts of yellow corn and oats developed typical rickets-like symptoms. Such animals showed stiffness and soreness in the legs, enlargements at the metatarsal, metacarpel, knee and hock joints, and stood humped with knees bent forward. During the latter part of the feeding period there was a tendency toward a chronic bloated condition with lack of appetite for grain and a consequent decrease in rate of growth and gain in weight. There was a distinct tendency for the calcium and to some extent for the inorganic phosphorus of the blood serum to decline with the onset of the deficiency. They pointed out that X-ray photographs of various bones clearly showed a "rachitic" condition. The epiphyseal lines were very irregular and displayed inferior calcification. A beaded area and irregular epiphyseal lines were revealed at the costo-chondral junction of the ribs. The low ash content of the moisture- and fat-free bones as contrasted with those from normal animals is further evidence of poor calcification of the skeleton. They found that the "rickets" was prevented or cured by feeding cod liver oil, viosterol, or high-quality sun-cured hay. The same results were secured by daily exposure of the calves, or the feed consumed by the calves, to ultra-violet irradiation. Parallel rat-feeding trials on rachitogenic rations, dehydrated hays, and sun-cured hays yielded substantially similar results.

Theiler (1934) states that "rickets is often found in calves on phosphorus deficient pastures. Under the ranching conditions of South Africa the calves are weaned at the age of about nine months and by that time calves of osteomalacic mothers are certainly inferior in weight and size to calves weaned from mothers receiving a supplement of phosphate. The explanation, however, is not congenital inferiority, but simply that their growth requirements are not covered by the quantity of milk obtained from the mothers subsisting on the phosphorus-deficient pasture, and that they themselves have to make up too large a proportion of their food at too early an age from the same deficient veld. We noticed rickets particularly in calves which had lost their dams early in life and which received an inadequate share of milk from foster mothers."

Theiler also expresses the opinion that altho normal mineral metabolism in all animals depends upon the presence of proper amounts of the minerals calcium and phosphorus and the essential vitamins, all animals do not necessarily react alike to a deficiency in any one or more of these factors. He states "some species react more readily to one deficiency than another, whilst in other species the same individual may react in two different ways at the same time. Different pathological pictures appear to result from identical causes, and the same pathological picture may be presented by diseases which from the aetiological point of view are different."

EXPERIMENTAL

The foregoing review and field surveys suggest that some form of nutritional deficiency is responsible for the occurrence of the rickets-like disease observed in calves on farms. The deficiency appears to be calcium, phosphorus, or vitamin D, or a combination of two or more of these.

Several methods were followed in determining the relationship of these factors to the disease. To secure more complete information relating to the conditions under which the syndrome may occur, as well as to the manner in which it affects the animal during its progress, the disease was produced experimentally in a number of cases. The relationship of the minerals, calcium and phosphorus, and of vitamin D was also ascertained by the results obtained when different levels of intake of each of these possible factors were provided and by noting the effect of adding each of these as a supplement to the rations of affected animals.

Animals Used, Feeding and Management

Data from 25 animals are included in the study. Of these, five (E199, E202, E203, E204, E206) were calves in an affected condition obtained from farmers in various parts of Minnesota. Some of the others that were affected were animals that had been assigned to other experiments. Care was exercised to include only those that conformed in their plan of feeding and management with the requirements of the specific phase or phases of the study in which their data were used.

With two or three exceptions, all the animals were six months old or over when placed on experiment. All calves reared at the station were given normal care and treatment up to the time they were placed on experiment. All the animals, when indoors, were confined in box stalls of sufficient size to allow considerable freedom and comfort. A large lot, entirely free from vegetation, was provided for outdoor exercise. Wood shavings were used for bedding in the box stalls.

No definite rule was followed in the selection of feedstuffs, except in the cases in which hay was excluded from the ration. Consideration was given only to fulfilling the requirements of the feeding standard with respect to protein, net energy, and the plan of the experiment with regard to calcium, phosphorus, and vitamin D. Except as indicated, the rations generally included varying amounts of prairie hay, beet pulp, yellow corn, occasionally oats, barley, and starch, with sufficient corn gluten meal to supply the needed amount of protein. Complete records were kept of the kinds and amounts of feedstuffs consumed, the nutrient and mineral intakes, and blood analyses and weights of each animal during the period on experiment. These records are on file in the Division of Dairy Husbandry, and upon request will be loaned to responsible persons for study.

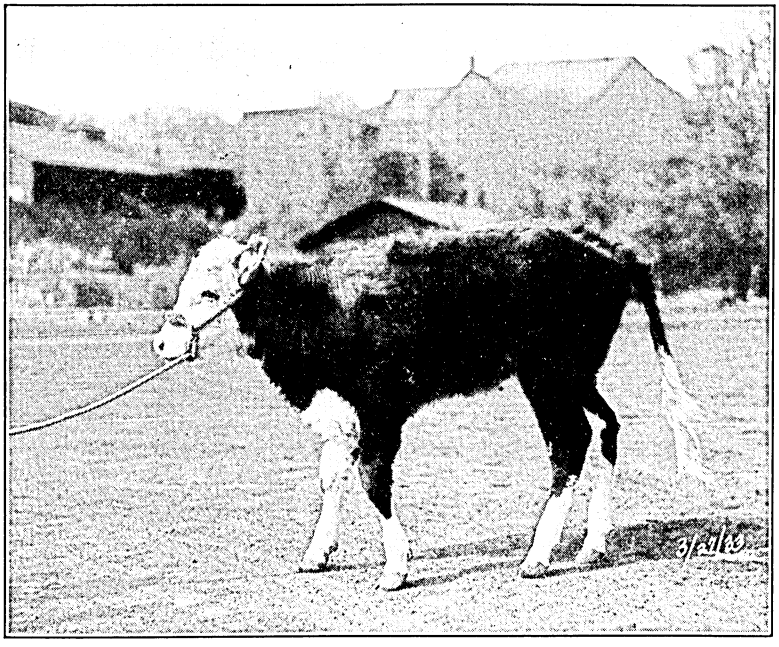

Fig. 1. E199 Soon After Her Arrival from the Farm
Note her rough, unkempt coat and decrepit appearance.

The weight of each animal was obtained under standard conditions once every 10 days, and every 30 days the weight was taken on three successive days. Observations regarding the outward appearance and general condition of each animal were made and recorded at frequent intervals. Brief abstracts of these observations are given as a part of the life history of each animal (see appendix). Determinations of the

calcium and the inorganic phosphorus content of the blood plasma were usually made at 30-day intervals or oftener, the determinations usually representing a composite of samples taken on three successive days. Post-mortem examinations were made of all animals that died or were slaughtered. Representative bone samples were obtained from some of them for chemical analysis.

Experimental Data

The data from the cases in which the disease was produced under experimental conditions are summarized in Table 1. The data indicating the efficiency of calcium, phosphorus, and vitamin D, respectively, in restoring affected animals to a normal condition are presented in Tables 2, 3, and 4. Table 5 summarizes the data from animals used but in which the syndrome did not develop. This table not only indicates the relationship to the disease of the several factors considered, but also suggests plans of feeding and management to be followed in combating it.

GENERAL DISCUSSION

The Relationship of Vitamin D and Calcium to the Disease

The experimental results summarized in Tables 1 to 5, inclusive, indicate that both the vitamin D supply and the calcium intake are involved in producing the disease as it occurs in calves on farms. The influence of vitamin D as a factor is established especially by the fact that in no instance was the disease produced except when there was a limited or deficient supply of vitamin D (Table 1). Its importance is also indicated by the fact that in extreme cases like those of the last experimental periods of E185, E194, and E196, as shown in Table 4, when the addition of calcium (Table 2) had previously failed to bring about complete recovery, the addition of a supply of vitamin D brought about complete recovery or at least great improvement in all cases. These results seemed to indicate that vitamin D is the more important of the two factors. The results with E146 also are in agreement with this. The data in Table 4 show that this animal on an extremely low calcium intake was completely restored to normal following the addition of the cod liver oil supplement. She remained normal in appearance and made excellent growth for about seven months after the cod liver oil feeding was discontinued, even tho she received less than 2.5 grams calcium daily during this period (unpublished data). At the end of that time (March), the characteristic symptoms began to reappear, but, with more hours of exposure to sunshine during April, the symptoms disappeared without any change being made in the ration fed.

Table 1.—Animals in Which the Syndrome Was Produced Experimentally, Indicating the Conditions Under Which It Occurred and Its Effect on the Physical Condition of the Animal and on the Calcium and Inorganic Phosphorus Content of the Blood Plasma

No. of animal	Date of experimental period (inclusive)	Supplement provided daily	Hay provided daily	Sunshine provided daily	Approximate mineral intake daily per 1,000 lb. of weight		Vitamin D supply	Physical condition		Blood plasma analysis			
										Start of period		End of period	
					Ca	P		Start of period	End of period	Ca	Inorg. P	Ca	Inorg. P
		gm.	lb.	hr.	gm.	gm.				mgm. per cent	mgm. per cent	mgm. per cent	mgm. per cent
E146	11- 3-30 to 3- 6-31	0.0[a]	1.0[b]	Limited[c]	6.0	25.0	Limited	Normal	Stiff	10.75	7.54	10.86[d]	6.67
E159	6- 1-31 to 2-15-32	0.0	1-3.5[e]	" c	13.5	12.5	"	"	Very stiff	10.92	6.35	8.96	2.81
E162	7-31-31 to 3-14-32	0.0[f]	1.5-4[g]	" c	30.0	14.0	"	"	Stiff	12.00	4.71	9.09	3.33
E164	8-30-31 to 3-22-32	0.0	1.0[h]	" l	4.0	18.0	"	"	"	10.91	7.14	5.76	7.46
E166	8-30-31 to 1-10-32	0.0[j]	1.0[r]	" l	9.5	18.0	"	"	"	10.36	6.94	6.03	6.85
E168	10-16-31 to 5-13-32	0.0[k]	3-5[l]	" m	30.0[n]	15.0	"	"	Very stiff	9.68	6.75	8.74	3.82
E169	11-28-31 to 3-26-32	0.0	1-3[o]	" c	13.5	13.0	"	"	Stiff	10.33	7.21	8.62	5.03
E170	12-28-31 to 4-27-32	0.0	3.0	" p	15.0	14.5	"	"	"	10.39	6.89	7.48	5.49
E185	5-21-33 to 8- 9-33	0.0	0.0[q]	0.0	12.0	16.0	Very limited	"	Very stiff	9.90	5.29	8.37	3.95
E187	6-22-33 to 11- 3-33	$CaCO_3$	0.0	0.0	43.0	11.0	0.0	"	" "	10.92	4.65	7.94	3.45
E190	6-22-33 to 10- 7-33	$CaCO_3$	0.0	0.0	48.0	11.5	0.0	"	" "	11.47	3.67	9.07	3.56
E194	5-21-33 to 8- 9-33	0.0	0.0[s]	0.0	12.0	14.0	Very limited	"	Stiff	10.35	6.08	6.28	5.78
E196	5-21-33 to 8- 7-33	0.0	0.0[t]	0.0	25.0	28.0	" "	"	"	10.43	8.09	4.39	5.74
E197	5-21-33 to 8-15-33	0.0	0.0[t]	0.0	24.0	25.0	" "	"	"	10.75	6.69	5.19	6.58
E206	3-14-33 to 6- 6-33	0.0	0.0[u]	0.0	15.0	28.0	" "	"	"	10.21	6.94	7.31	3.45

[a] 10 gm. $NaH_2PO_4.H_2O$ fed daily up to 12-6-30.
[b] Timothy hay, 1 lb. daily, except that 2 lb. were fed 12-19-30 to 1-3-31 and from 2-12-31 to 3-6-31.
[c] 4-6 hours daily, spring, summer, and fall, less than 1 hour in winter.
[d] 8.16 mgm. per cent on 2/2-4.
[e] Prairie and timothy hay, only 1.5 lb. after 12-30-31.
[f] 12 gm. $CaCO_3$ daily from 10-16-31 to 11-14-31 and 8 gm. 2-12-32 to 3-9-32.
[g] Mostly prairie hay along with some alfalfa from 1-14-32 and on.
[h] 2 lb. timothy 11-28-31 to 3-10-32.
[i] No sunshine after 12-4-31.
[j] 25 gm $CaCO_3$ beginning 11-24-31.
[k] 37.5 gm. $CaCO_3$ beginning 3-23-32 along with 5 c.c. viosterol beginning 5-9-32.
[l] Mostly prairie with some alfalfa 1-14-32 to 2-9-32.
[m] Same as [e] but no sunshine after 3-15-32.
[n] 50 gm. after 3-23-32.
[o] Timothy and prairie up to 1-13-32 then prairie.
[p] Same as [e] but no sunshine after 3-26-32.
[q] Prairie hay reduced to 1 lb. on 6-7-33, discontinued entirely on 7-6-33.
[r] Timothy hay.
[s] 2 lb. prairie hay daily 5-21-33 to 7-6-33, no hay after 7-19-33.
[t] Approximately 1 lb. prairie hay daily up to 7-19-33.
[u] Prairie hay.

Table 2.—The Effect of Adding a Calcium Supplement to the Rations of Cattle Affected with the Syndrome

No. of animal	Date of experimental period (inclusive)	$CaCO_3$ provided daily (approx.)	Hay provided daily	Sunshine provided daily	Approximate mineral intake daily per 1,000 lb. of weight		Vitamin D supply	Physical condition		Blood plasma analysis			
								Start of period	End of period	Start of period		End of period	
					Ca	P				Ca	Inorg. P	Ca	Inorg. P
		gm.	lb.	hr.	gm.	gm.				mgm. per cent	mgm. per cent	mgm. per cent	mgm. per cent
E162	3-15-32 to 12-21-32	0.0[a]	10.0[b]	0.0[c]	50-175	25	Liberal	Stiff	Nearly normal	9.09	3.33	10.53	5.25
E164	3-23-32 to 8-31-32	125	1.0[d]	0.0	75	17	Limited	"	Normal	5.76	7.46	10.22	6.54
E166	1-11-32 to 4-14-32	100	1.0[d]	0.0	70	18	"	"	Nearly normal	6.03	6.85	10.38	5.57
E170	4-28-32 to 1-20-33	100	4-9[e]	0.0	85	14	Quite liberal	"	Normal	7.48	5.49	10.68	5.02
E199	3- 2-33 to 5-20-33	100	1.5[e]	0.0	135	20	Limited	Very stiff	Much improved	6.71	4.85	9.50	5.09
E202	3-20-33 to 10- 4-33	100	2.0[e]	0.0	120	21	"	" "	"	8.51	4.28	10.27	4.56
E203	3-21-33 to 10- 4-33	100	2.0[e]	0.0	110	20	"	" "	"	7.23	4.18	8.81	3.66
E204	3-10-33 to 10- 4-33	100	2.0[e]	0.0	110	21	"	" "	"	7.67	3.36	9.02	3.50
E206	6- 7-33 to 10-17-33	75	2.0[e]	0.0	90	20	"	Stiff	"	7.31	3.45	8.71	7.09
E185	8-10-33 to 9-25-33	100	0.0	0.0	80	14	0.0	"	Extremely decrepit	8.37	3.95	8.41	3.13
E194	8-10-33 to 9-25-33	100	0.0	0.0	70	12	0.0	"	Very stiff	6.28	5.78	8.30	3.40
E196	8- 8-33 to 10-15-33	75	0.0	0.0	90	20	0.0	"	Very bad condition	4.39	5.74	6.33	4.66
E197	8-16-33 to 10-14-33	75	0.0	0.0	90	20	0.0	"	Died in convulsion	5.19	6.58	4.98	5.63

[a] 5 c.c. viosterol daily from 10-6-32 to 11-21-32, then 2 c.c.
[b] Alfalfa hay.
[c] None until 9-1-32, then about 5 hours daily.
[d] Timothy hay.
[e] Prairie hay.

Table 3.—The Effect of Adding a Phosphorus Supplement to the Rations of Cattle Affected with the Syndrome

No. of animal	Date of experimental period (inclusive)	Amount of $NaH_2PO_4.H_2O$ provided daily	Prairie hay provided daily	Sunshine provided daily	Approximate mineral intake daily per 1,000 lb. of weight		Vitamin D supply	Physical condition		Blood plasma analysis			
										Start of period		End of period	
					Ca	P		Start of period	End of period	Ca	Inorg. P	Ca	Inorg. P
		gm.	lb.	hr.	gm.	gm.				mgm. per cent	mgm. per cent	mgm. per cent	mgm. per cent
E202	3-10-33 to 3-19-33	35[a]	2.0	0.0	16	60	Limited	Stiff	Collapsed	9.06	3.69	8.51	4.28
E203	3-15-33 to 3-20-33	40	2.0	0.0	15	45	"	"	"	7.62	4.24	7.23	4.18
E187	11- 4-33 to 11-22-33	40[b]	0.0	0.0	40	26	"	"	Near collapse	7.94	3.45	6.12	5.26

[a] His ration also included about 1.25 lb. wheat bran daily.
[b] Also 55 gm. $CaCO_3$ daily.

Table 4.—The Effect of Adding Vitamin D Supplements to the Rations of Cattle Affected with the Syndrome

No. of animal	Date of experimental period (inclusive)	Supplements provided daily	Hay provided daily	Sunshine provided daily	Approximate mineral intake daily per 1,000 lb. of weight		Vitamin D supply	Physical condition		Blood plasma analysis			
										Start of period		End of period	
					Ca	P		Start of period	End of period	Ca	Inorg. P	Ca	Inorg. P
		gm.	lb.	hr.	gm.	gm.				mgm. per cent	mgm. per cent	mgm. per cent	mgm. per cent
E146	3- 7-31 to 8- 6-31	Cod liver oil[a]	2.0[b]	2-6	5.0	23.5	Liberal	Stiff	Normal	10.86	6.67	10.68	7.78
E166	4-15-32 to 5- 4-32	Sunshine	1.0[c]	3-5	85.0	20.0	"	Nearly normal	"	9.45[e]	5.78[e]	10.38[f]	5.57[f]
E169	3-27-32 to 1-20-33	Viosterol[d]	5.0	0.0	15.0	13.0	"	Stiff	"	8.62	5.03	9.88	7.43
E185	9-26-33 to 10-17-33	$CaCO_3$ and Viosterol[g]	0.0	0.0	56.0	9.0	"	Very stiff	Much improved	8.41	3.13	11.82	4.08
E187	11-23-33 to 3- 5-34	$CaCO_3$ and Viosterol[h]	4.0[i]	0.0	65.0	16.0	"	" "	Improved	6.12	5.26	9.56[o]	8.73[o]
E190	10- 8-33 to 1-15-34	$CaCO_3$ and Sunshine[j]	0.0[k]	1-3	45.0	15.0	"	" "	Much improved	9.07	3.56	10.88	6.02
E194	9-26-33 to 11- 2-33	$CaCO_3$ and Viosterol[l]	0.0	0.0	75.0	11.0	"	Stiff	Normal	8.30	3.40	11.11	6.41
E196	10-16-33 to 1-15-34	$CaCO_3$[m]	0.0[n]	1-3	90.0	28.0	"	"	Nearly normal	6.33	4.66	10.57	9.35

[a] 100 c.c. daily.
[b] Timothy, only 1 lb. after 5-4-31.
[c] Timothy hay.
[d] 5 c.c. daily from 3-27-32 to 5-15-32, then 2 c.c.
[e] Analysis made 4/1-3.
[f] Analysis made 4/19-21.
[g] 50 to 100 gm. $CaCO_3$ and 5 c.c. viosterol daily.
[h] 55 gm. $CaCO_3$ daily. 5 c.c. viosterol injected into jugular vein daily 11-22-33 to 11-30-33, inclusive, after this 5 to 3 c.c. were fed daily to 2-8-34, after which it was discontinued.
[i] Alfalfa hay. No hay fed from 1-16-34 to 2-2-34, 5 to 7 lb. daily after that.
[j] 55-60 gm. $CaCO_3$ daily.
[k] 5 lb. alfalfa hay daily 12-17-33 to 1-8-34.
[l] 100 gm. $CaCO_3$ and 5 c.c. viosterol daily.
[m] 100 gm. daily.
[n] A total of 8 lb. prairie hay was fed during period 10-14-33 to 10-17-33 inclusive.
[o] Analysis made 2/7-9.

Table 5.—Experimental Animals in Which the Syndrome Did Not Develop, Indicating the Conditions Required To Prevent Its Occurrence, i.e., an Adequate Supply of Both Calcium and Phosphorus and Some Source of Vitamin D (hay, sunshine, viosterol)

No. of animal	Date of experimental period (inclusive)	Supplement provided daily	Hay provided daily	Sunshine provided daily	Approximate mineral intake daily per 1,000 lb. of weight		Vitamin D supply	Physical condition		Blood plasma analysis			
										Start of period		End of period	
					Ca	P		Start of period	End of period	Ca	Inorg. P	Ca	Inorg. P
		gm.	lb.	hr.	gm.	gm.				mgm. per cent	mgm. per cent	mgm. per cent	mgm. per cent
E161	7- 1-31 to 10-31-32	$CaCO_3$[a]	1-8[b]	0.0[c]	120-230	10-20	Liberal	Normal	Normal	12.22[d]	3.56[d]	10.43	5.90
E180	8-24-32 to 6-23-33	0.0[e]	5-6[f]	0.0	28	15	"	"	"	9.94	8.73	10.04	7.75
E181	8-24-32 to 5- 8-33	0.0[g]	3-5[f]	2-4	26	15	"	"	"	10.11	8.44	10.76	6.29
E182	8-24-32 to 6-23-33	Viosterol[h]	3-7[f]	0.0	28	15	"	"	"	10.82	5.99	10.78	6.96
E185	11-22-32 to 5-20-33	0.0	3-4.5[f]	0.0	25	27	Quite liberal	"	"	10.64	7.43	9.90	5.29
E186	12-22-32 to 5-20-33	0.0	3-4.5[f]	2-4	30	27	Liberal	"	"	10.22	7.75	9.74	8.85
E186	5-21-33 to 10-17-33	0.0	0.0[i]	6-8	12	16	"	"	"	9.74	8.85	10.06	7.43
E187	1-21-33 to 6-21-33	$CaCO_3$	3-6[f]	0.0	48	17	"	"	"	10.14	7.81	11.10	4.67
E190	1-21-33 to 6-21-33	$CaCO_3$	3.5-5[f]	0.0	100	20	"	"	"	10.35	7.54	11.47	3.67
E194	1-21-33 to 5-20-33	0.0	4-5[f]	0.0	25	17	"	"	"	10.60	6.37	10.35	6.08
E198	5-21-33 to 12-16-33	0.0	1-8[f]	0.0	30	22	Adequate	"	"	10.48	6.54	10.13	5.95

[a] Variable amounts.
[b] Prairie and alfalfa.
[c] Not exposed to direct sunshine after 3-14-32.
[d] Analysis made 8/3-5.
[e] 6 to 9 gm. $CaCO_3$ from 11-3-32 to 1-20-33 and 8 to 14 gm. 3-22-33 to 6-23-33 4 to 12 gm. $NaH_2PO_4.H_2O$ from 4-21-33 to 6-23-33.
[f] Prairie hay.
[g] 2 to 4 gm. $CaCO_3$ from 9-19-32 to 1-20-33.
[h] 2 c.c. viosterol daily. 6 to 9 gm. $CaCO_3$ from 11-5-32 to 12-21-32 and 8 to 14 gm. from 3-22-33 to 6-23-33. 8 gm. $NaH_2PO_4.H_2O$ from 4-21-33 to 6-23-33.
[i] Hay (prairie) reduced to 1 lb. daily 6-8-33 and discontinued entirely 7-6-33.

It is probable that the disease had caused permanent injury in those animals in which complete recovery was not attained, even after an adequate supply of both vitamin D and calcium had been provided, as in the cases of E162 (Tables 1 and 2) and E187 (Tables 5, 1, 3, and 4).

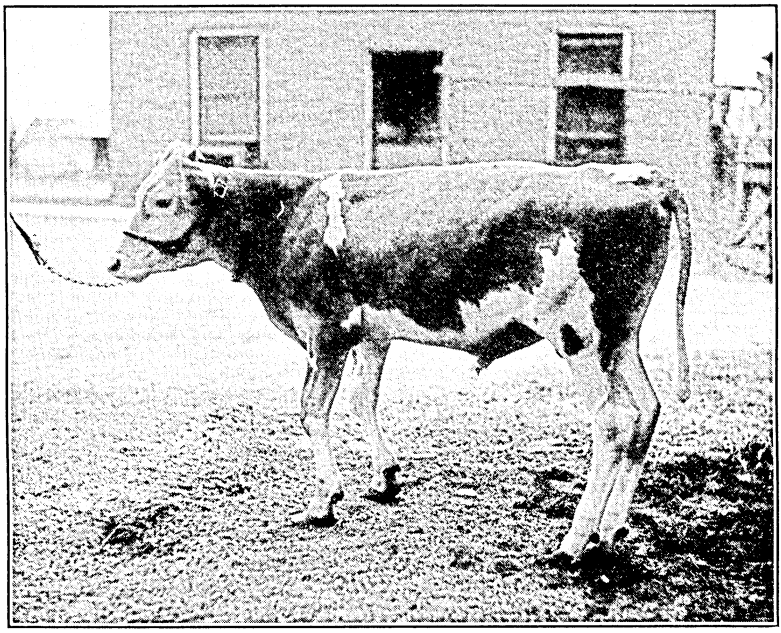

Fig. 2. Physical Evidence of the Presence of Syndrome as Exhibited by E206 After Being Fed a Low Calcium Ration Along with a Very Inadequate Supply of Vitamin D

The influence of calcium as a factor is apparent in that there appears to be a more or less direct relationship between the level of the calcium intake and the onset of the disease. The cases of E161 (Table 5) and E170 (Tables 1 and 2), in particular, suggest this. It is significant that E161, on a calcium intake of from 120 to 230 grams daily per 1,000 pounds liveweight, failed to show any symptoms of the syndrome during the entire 16 months she was on experiment, while E170 (see Table 1), under practically the same conditions of feeding and management but on a calcium intake less than one-tenth as high, developed the disease within four or five months. E170 was later (Table 2) restored to complete normal physical condition when provided with a calcium supplement.

A similar effect of the level of the calcium intake on the development of the syndrome is to be found in the case of the group of affected calves E199, E202, E203, and E204 (Table 2) that were obtained from

farmers. These animals failed to show any improvement during the first week or more after their arrival when fed a ration low in calcium, similar to that received on the farm. Later, however, when additional calcium (Table 2) was provided, they made marked progress toward recovery, as indicated by improvement in their physical condition and the return of the mineral content of the blood to normal. It is apparent from this that the development of the disease might have been prevented in these animals while on the farm by providing them with a more liberal intake of calcium.

The influence of calcium is shown further by the fact that in all cases in which affected animals were provided with a calcium supplement, as shown in Table 2, some improvement was indicated by a rise in the blood calcium. The life histories of these animals indicate that in most cases it was followed by improvement in physical condition as well. It is true that the extent of improvement varied largely according to the degree of deficiency of vitamin D; nevertheless, even in such extreme cases as those of E185, E196, and E197 (Table 2), on rations almost if not completely devoid of vitamin D, improvement in either the blood calcium or physical condition or in both was evident. In the case of E170, as already stated, complete restoration to normal apparently followed the addition of the calcium supplement to the ration.

From these facts, it is apparent that a high calcium intake either exerts a sparing effect on the amount of vitamin D required, or vice versa. Wallis, Palmer, and Gullickson (1935) have definitely demonstrated that a low vitamin D supply increases the calcium requirement by decreasing the percentage of mineral retained by the animal. The final result is the same in either case.

On this basis it is evident that the disease may, and probably does, occur in calves on farms, under various conditions, and perhaps in various forms, depending on the relative degree of deficiency of the respective factors, as well as on the individuality of the animals concerned. E146 and E164 (Table 1) are good examples of affected animals on rations extremely deficient in calcium, but providing a fairly liberal amount of vitamin D. Examples of the opposite extreme are such animals as E187, E190, E196, and E197 (Table 1) and E185, E194, E196, and E197 (Table 2) that received rations almost, if not completely, devoid of vitamin D but a fairly liberal supply of calcium. All the other experimental animals that were affected with the disease represent various forms of the syndrome between these two extremes.

It is apparent that the disease may have a multiple etiology depending on environmental conditions. The time required for its development as well as its degree of severity appears to be determined by the

extent of the deficiency of the factors considered and, also, by the animal's requirements of them. Regarding the latter point, the evidence from various sources indicates that young, rapidly growing animals have a proportionately higher mineral and vitamin D requirement and are therefore more prone to be affected by the syndrome than are more mature animals under similar conditions of feeding and management. This is what one would expect.

Phosphorus as a Factor

As can be observed in Tables 1, 2, 4, and 5, there is a considerable range in phosphorus intake among both the affected and non-affected animals. There is, however, no evidence to indicate that a low intake of phosphorus is a primary etiological factor, altho it may eventually complicate the disease, as it apparently did in the case of E159 and E190 (Table 1). On the other hand, the experimental evidence from E187, E202, and E203, as summarized in Table 3, indicates a possible harmful effect resulting from providing affected animals with a too liberal supply of this element. These animals were provided with a phosphorus supplement, in the form $NaH_2PO_4.H_2O$, at a time when they were afflicted with the disease. When this was done, they became so stiff and decrepit that they could hardly stand. When a generous amount of calcium carbonate replaced the phosphorus supplement in the rations of E202 and E203 the animals began to improve immediately (Table 2).

It is difficult to account for the collapse of these three animals following the addition of the phosphorus supplement to their rations. The fact that E202 and E203 were probably suffering from a deficiency of both vitamin D and calcium at the time the supplement was provided suggests that the added phosphorus may have changed the calcium-phosphorus ratio to such an extent as to augment further the existing calcium shortage due to the metabolic relationships established. Mineral balance trials would have been required to determine this. The situation with respect to E187 is only slightly different. The ration fed this animal contained a liberal amount of calcium but was completely devoid of both hay and vitamin D. Under these conditions it is probable that the amount of calcium retained was no greater than in each of the other two and similar results would therefore be expected in all three cases.

The addition of phosphorus to the ration resulted in an improvement in the appetites of E202 and E203 for hay. It is probable that if these animals, while receiving the phosphorus supplement, had been allowed to consume all the hay they wanted, they would have recovered normal physical well-being. This, however, would not have been due to the additional phosphorus provided, but to the increased amounts of

calcium and vitamin D supplied by the greater hay consumption. It is apparent that under farm conditions this plan of procedure might prove satisfactory. It is equally apparent why the disease is frequently found in cattle on farms located in phosphorus-deficient regions, for, as has been well established, a lack of appetite for hay invariably follows a deficiency of phosphorus in the ration.

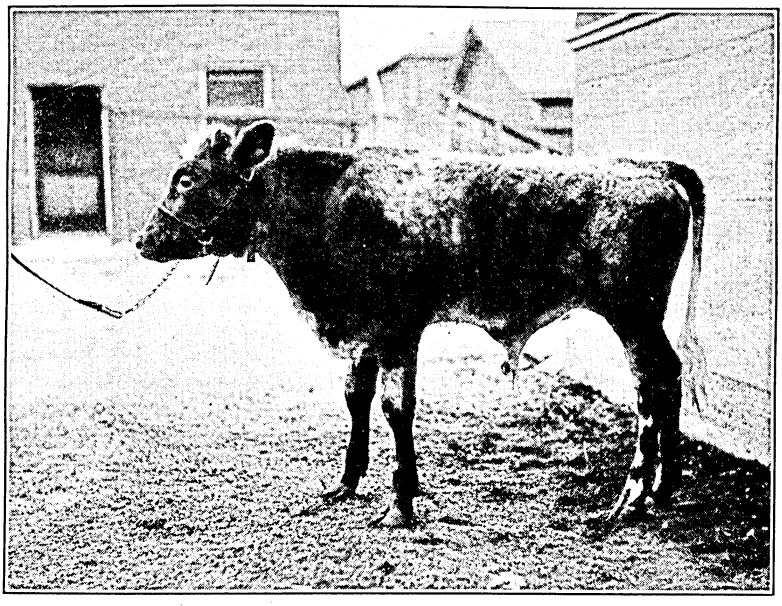

Fig. 3. E204 Near the End of His Experimental Period, After Receiving a Calcium Supplement to the Ration

Note his nearly normal appearance. This animal was hardly able to stand alone at the time the calcium supplement was first added.

Hay as a Source of the Protective Factors

Much evidence has been presented indicating the importance of hay or roughage in the rations of cattle. The experimental data also indicate the value of hay as a protection against the development of the disease with which this study is concerned.

The data from the three animals E196, E197 (Tables 1 and 2), and E198 (Table 5) are especially significant. These calves when started on experiment, May 21, 1933, were all fed similar rations except E198 which received an adequate hay intake. The others received only about one pound of prairie hay daily during the first 60 days, after which dry molasses beet pulp was provided as the only source of bulky feed. From the data recorded in Tables 1 and 2, and their life history in the appendix, it will be observed that E196 and E197, on the low

hay and hayless rations, suffered almost complete collapse—as indicated by attacks of fits, stiffness, bent knees, and almost constant bloating, within less than 60 days after they were started on experiment. E198 (Table 5), on the other hand, receiving prairie hay ad libitum, remained normal in outward appearance. It is significant to note, however, that while E198 did not exhibit any definite visible symptoms of the disease during this period, her blood calcium remained between 7 and 8 mgm. per 100 c.c. plasma for three consecutive months of the time, which was much lower than that of many animals that were affected. These data are shown in Table 6. This fact suggests that the rather limited supply of vitamin D or other factor in the hay fed was able to prevent the development of the disease in the form in which it is usually recognized, but there was insufficient intake and/or absorption of calcium, and possibly of other essential factors, to maintain the blood calcium at a normal level. In other words, this animal was in a state of acalcicosis during a considerable portion of the period. It is probable also that this animal was on the borderline of showing the characteristic symptoms of the disease during the first part of the experiment, as the blood calcium began to rise toward normal when the hay consumption increased to six pounds daily. These results, when considered as a whole, furnish, in part, the basis for the statement previously made that the disease probably has a multiple etiology and the form of its manifestation depends upon the relative degree of deficiency of the factors involved. The results from E196 and E197 during the period of complete lack of vitamin D also furnish evidence in support of this. They are also of importance in indicating certain facts regarding the nature and characteristics of the disease. Following the development of the disease in E196, after several months on experiment, this animal showed some improvement when $CaCO_3$ (Table 2) was added to her ration, but this was only temporary. When turned out-of-doors in mid-October, with no further change in ration, recovery was practically complete by January 15. It seems evident that the small amount of active ultra-violet rays received at that time of the year and the ample supply of calcium provided were responsible for recovery. E197, likewise, showed slight temporary improvement following the addition of $CaCO_3$ to her vitamin D deficient ration, but died before vitamin D therapy could be begun. From a study of Tables 7 and 8 it will also be observed that these animals did not develop anorexia to a very marked degree. This was in marked contrast with other animals affected, the data for which are not included in this bulletin. This result may have been due to the stronger growth impulse of these younger animals, causing a more persistent desire for food. It will also be noted that both of these animals

suffered from several attacks of fits, E197 succumbing to such an attack on October 14, 1933. The other, older animals affected seldom, if ever, suffered from such attacks. This suggests that the more extreme form of the disease appears mostly in younger animals and that the most susceptible period in the life of the calf is the time when growth is most rapid.

That some factor other than the increased calcium intake, which followed as a natural course the increase in hay consumption, by E198, was responsible for her normal appearance throughout is indicated by the fact that the addition of a calcium supplement to the rations of E196 and E197 (Table 2) when they were seriously affected produced only a slight and very temporary improvement in their condition. The same was true when a calcium supplement was added to the hayless diets of E185 and E194 during the period from August 10 to September 26. These results are in accord with those obtained by Bechdel and associates (1933) in which they found that adding the ash from two and one-half pounds of sun-cured alfalfa hay (sufficient to protect calves against rickets) to the basal rachitogenic ration failed to prevent the rickets-like symptoms from developing. When calcium was added to the rations of the group of affected animals (E199, E202, E203, E204, and E206) from the farm, they all began to improve immediately. The significant fact is that they were all receiving some hay in their rations. It will also be noted that all of the animals except E186, second period, listed in Table 5, which did not develop any symptoms of the disease received an abundance of hay in addition to ample calcium. Some also received viosterol or sunshine. E186 was evidently protected in part by her previous stores of calcium and vitamin D and by the many hours of effective sunshine received.

These results are significant, not only in indicating that prairie hay is a source of protective factors, but in explaining why in the same herd, young calves, which vary widely in their ability and in their desire to consume roughage, apparently show great variation in susceptibility to the disease.

Further evidence of the value of hay as a protective agent is offered in the experiment conducted by Gullickson and Eckles (1927), previously referred to. Two Holstein heifers were kept in complete darkness during a period of nearly two years, during which time their rations included a liberal supply of good-quality timothy hay along with a grain mixture. These animals remained normal throughout. The hay must have been the chief, if not the only, source of vitamin D. It is probable that a similar explanation might apply to other experiments which have been interpreted as indicating that cattle require little or no vitamin D.

Samples of prairie hay have been tested for vitamin D content by Wallis (1934) (see Wallis, Palmer, and Gullickson 1935) and found to contain from 15 to 30 Steenbock units per pound. Calves consuming 4 to 6 pounds of such hay per day, in addition to adequate calcium intake, did not develop any symptoms of the rachitic-like disease. How much less would have protected them is not certain, but the experimental data in Table 5 suggest that the vitamin D requirement may be less than 100 Steenbock units per day when the intake of calcium and phosphorus is adequate.

In considering the value of hay in the ration, it is worth while to inquire as to the possibility of its containing some other, still unknown factor or factors essential to the health and well-being of cattle. This query is suggested by the disastrous results reported invariably to follow the feeding of calves on rations devoid of hay. In some of these trials, the addition of various mineral supplements, together with cod liver oil, brought varying degrees of relief but if some form of hay was not included in the ration normal development seldom if ever took place. For this reason, it is pertinent to inquire whether or not a calf can be grown to maturity on a hayless ration even if provided with an adequate supply of the minerals and vitamins known to be required. This has not yet been demonstrated conclusively; in fact a study conducted by Bennett (1932) at this station strongly suggests the indispensability of hay to the normal well-being of dairy calves.

EFFECTS OF THE DISEASE

Rate of Gain in Weight

One of the most important effects of the rickets-like disease in cattle from the standpoint of the livestock raiser, is its effect on the rate of growth and gain in weight. Figures 4, 5, and 6, showing the growth, nutrient and mineral intake, together with the mineral content of the blood of three representative animals afflicted with the disease, are evidence that almost invariably the rate of gain was retarded. Other workers, notably Rupel, Bohstedt, and Hart (1933), have also noted this tendency of calves on vitamin D deficient rations to cease gaining in weight and have interpreted it as a direct effect of the vitamin D deficiency. The data in the present study, however, suggest that the loss in appetite, which invariably develops in affected animals, is an important cause of the decrease in rate of gain. Whether or not the disease has any effect on the efficiency of utilization of nutrients cannot be satisfactorily determined from the available data. It is reasonable to suppose, however, that any circumstances able to produce the

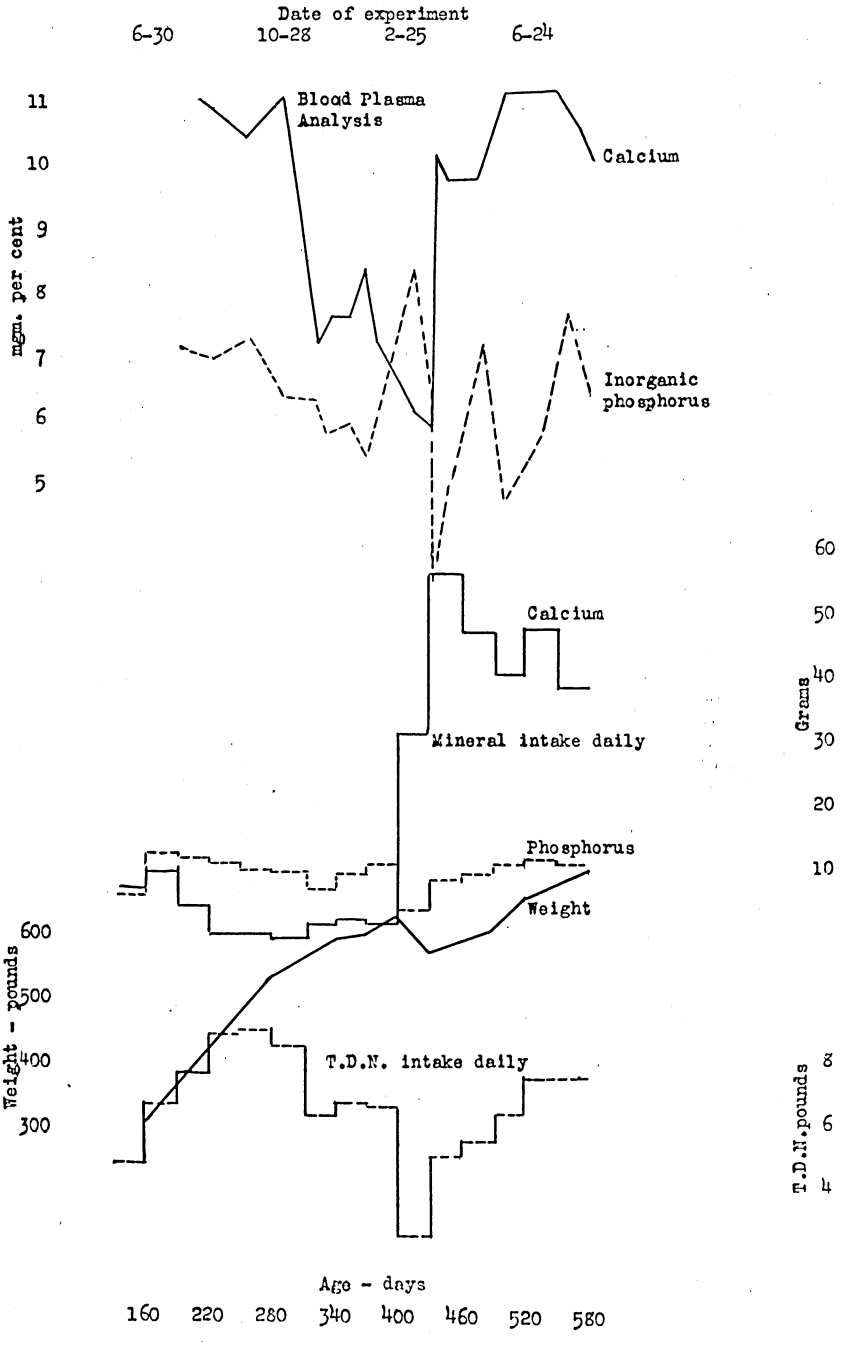

Fig. 4. The Growth, Nutrient and Mineral Intake, and Calcium and Inorganic Phosphorus Content of Blood Plasma of E164 During Period of Experiment

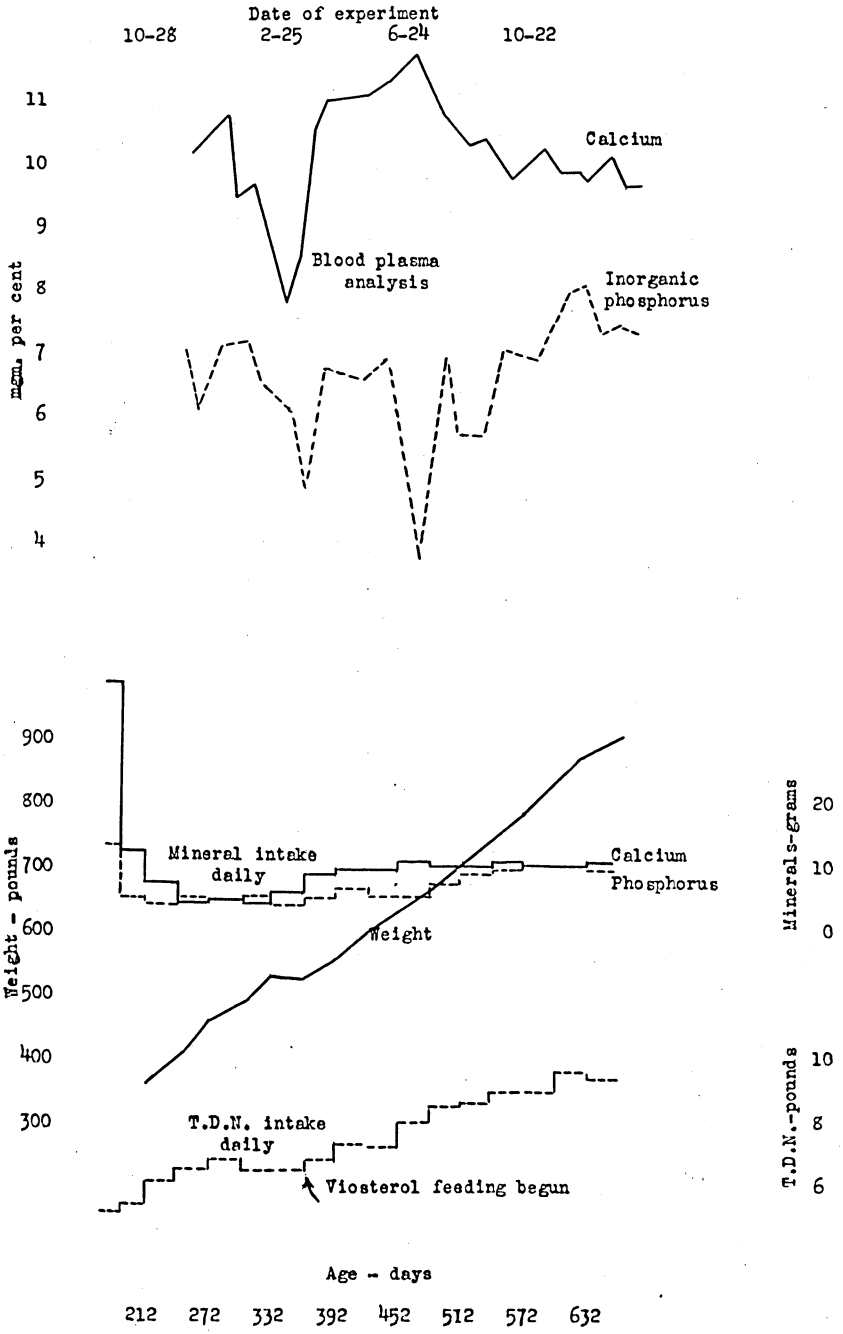

Fig. 5. The Growth, Nutrient and Mineral Intake, and Calcium and Inorganic Phosphorus Content of Blood Plasma of E169 During Period of Experiment

various forms of decrepitude observed may also be expected to affect the efficiency with which the nutrients are utilized. Digestive disturbances such as bloat, diarrhea, and constipation, which frequently accompany the disease, support this conclusion. Murray (1912) stated that bloat can be expected to occur whenever the digestive process is imperfectly performed and the food in the paunch ferments with the consequent production of gas.

The Calcium and Inorganic Phosphorus of the Blood

Rupel, Bohstedt, and Hart (1933) report that the inorganic phosphorus in blood of calves on diets deficient in vitamin D showed a marked and continued decline. They state: "It appears that a decline in the level of blood phosphorus in calves fed on rations as used in our experiments may be taken as one of the first evidences of the onset of rickets. We do not know that it is an infallible guide but find no disagreement with this interpretation among our data."

Bechdel and associates (1933), on the other hand, report that the blood calcium became abnormally low within a short time after the animal was placed on experiment and continued low throughout, while the inorganic phosphorus only tended to go down after a considerable period of time and then in most cases only slightly.

Huffman (1934) states that, in vitamin D deficiency, three types of blood pictures are observed: (1) normal blood calcium, low inorganic phosphorus (identical with low-phosphorus rickets), (2) low blood calcium, normal inorganic phosphorus, and (3) low blood calcium and low inorganic phosphorus.

Theiler (1934) concluded from his study of the osteodystrophic diseases of domesticated animals that "the normal function of the mineral metabolism of an animal depends upon the harmonious interaction of three dietary factors: vitamin, calcium and phosphorus. The absence of any one of these might be expected to have the same result in all animals, but in practice this does not seem to be the case. Some species react more readily to one deficiency than another whilst in other species the same individual may react in two different ways at the same time." Theiler further concludes that the inevitable pathological changes in the bones may vary according to age, species of animal, and mode of life. It appears from the experiments cited that similar variations occur in the calcium and phosphate content of the blood.

In considering our own experimental results, from the standpoint of the effect on the mineral content of the blood, it will be observed in all cases, excepting perhaps those of the animals definitely on phosphorus-deficient rations, that it was the calcium rather than the inorganic phos-

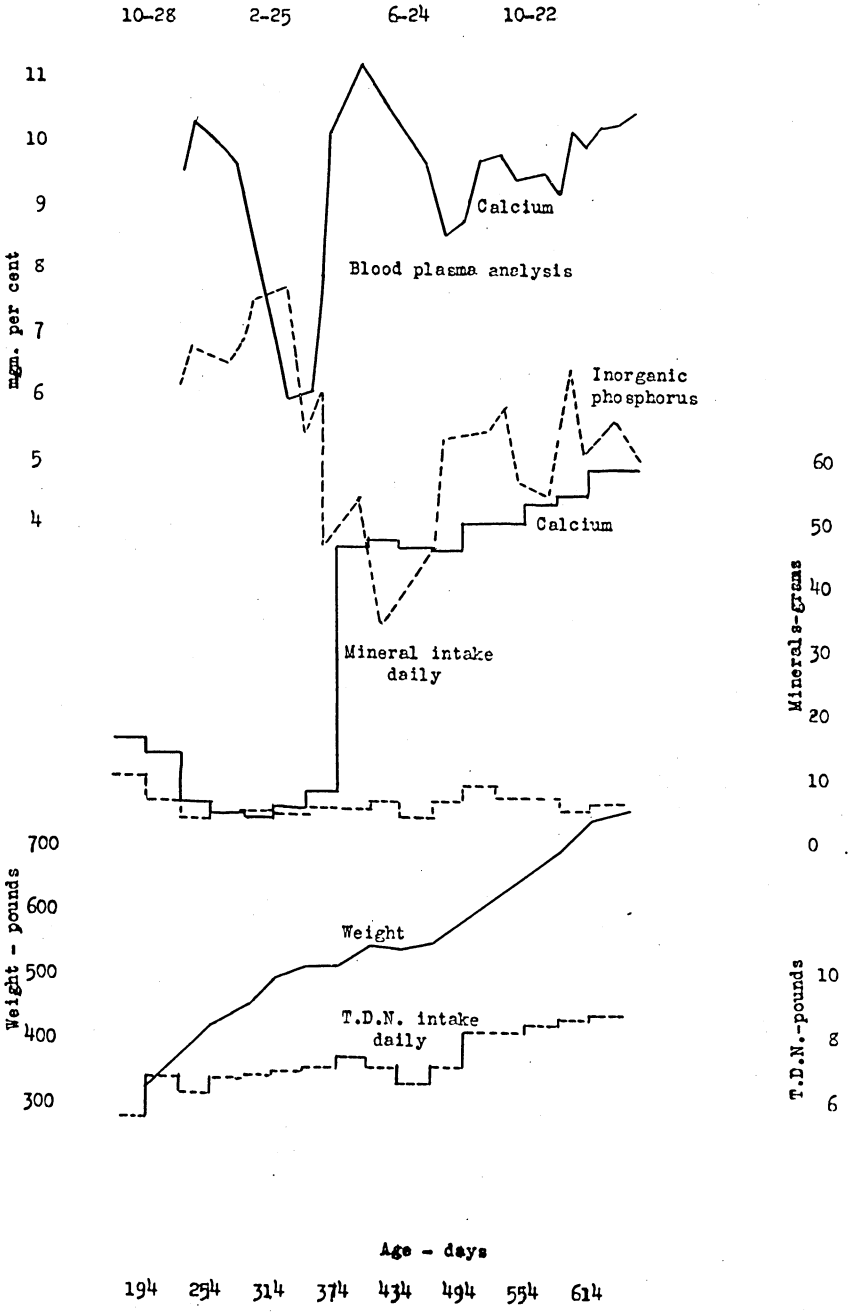

Fig. 6. The Growth, Nutrient and Mineral Intake, and Calcium and Inorganic Phosphorus Content of Blood Plasma of E170 During Period of Experiment

phorus of the blood plasma which showed the first tendency to decline with the onset of the disease. It has already been pointed out that blood samples obtained from affected cattle on farms also were usually low in calcium.

It is significant to note, however, from the results with E198 (Tables 5 and 6), that a drop in the blood calcium does not necessarily presage the development of the disease in the form ordinarily recognized. Also, it is apparent from the results with E190 (Table 1) and E185 (Table 2) that the syndrome may appear even when the blood calcium level is only slightly below normal. In this connection, it will be noted that both E185 and E190 during the periods indicated were provided with a fairly liberal intake of calcium but no vitamin D, whereas E198 on the average consumed enough hay to provide a fairly liberal amount of vitamin D with only a moderate amount of calcium. It is evident that vitamine D may be a more potent prophylactic agent than calcium in the prevention of the clinical symptoms of this disease.

Table 6.—Blood Plasma Composition of E198, E196, and E197 at Intervals During the Experimental Period

Date	E198		E196		E197	
	Ca	Inorg. P	Ca	Inorg. P	Ca	Inorg. P
	mgm. per 100 c.c.		mgm. per 100 c.c.		mgm. per 100 c.c.	
1933						
5/26-28	10.48	6.54	10.43	8.09	10.75	6.69
6/26-28	9.78	6.62	9.73	8.06	10.04	7.63
8/9	7.64	9.17	4.39	5.74	6.28	5.13
8/14-16	7.27	8.47	6.48	4.65	5.19	6.58
8/28-30	7.86	6.67	6.81	4.53	8.28	2.73
9/12-14	7.79	7.14	6.44	4.02	6.85	3.27
10/2-4	7.10	7.81	5.50	5.73	5.91	4.67
10/11-13	8.40	6.62	6.33	4.66	4.98[a]	5.63
11/13-15	9.91	7.69	10.78	9.08	...	...
12/11-13	10.13	5.95	10.76	8.97	...	...
1934						
1/9-11	....	...	10.57	9.35	...	...

[a] Died in tetany convulsion October 14.

It will be observed that almost invariably in the animals which became affected with the syndrome in our experiments, as typified in Figures 4, 5, and 6, the downward trend in the blood calcium was followed by a drop in the inorganic phosphorus. This latter effect, however, does not appear to be quite as marked as that of the calcium and seems to occur only if the environmental conditions that produced the disease are allowed to continue over a sufficient period of time. The receding of the inorganic phosphorus of the blood is probably a secondary development, resulting from the decrease in appetite for concentrates, which invariably develops as the disease progresses.

Table 7.—E196, Data Showing Age, Weight, Feedstuffs Consumed, and Daily Nutrient and Mineral Intake During Experimental Period

Date, end of period	Age	Weight	Feedstuffs consumed							Nutrient intake daily		Mineral intake daily	
			Skim-milk	Prairie hay	Corn	Oats	Corn gluten meal	Dry beet pulp	$CaCO_3$	Crude protein	Total digestible nutrients	Ca	P
	days	lb.	lb.	lb.	lb.	lb.	lb.	lb.	gm.	lb.	lb.	gm.	gm.
6-19-33	144	271	295	33.0	69.0	...	36.0	...	...	0.95	4.20	8.14	8.36
7-19-33	174	318	99	31.0	56.5	50.6	53.6	26.5	...	1.12	5.50	7.50	8.51
8- 7-33	193	346	...	...	60.7	49.5	22.9	26.2	...	0.99	6.30	5.25	8.69
8-18-33	204	333	...	...	26.0	9.5	8.5	10.5	715	0.58	3.80	29.25	4.79
9-17-33	234	376	Wheat bran	...	80.2	63.0	41.0	42.8	2,250	0.96	5.70	35.18	7.73
10-17-33	264	398	12.0	8.0	100.5	32.5	37.3	40.0	2,163	0.92	5.72	34.58	9.40
11-16-33	294	435	14.0	...	56.0	112.3	28.0	42.0	2,140	0.97	6.09	34.45	11.49
12-16-33	324	475	15.0	...	75.0	120.0	30.0	...	2,850	1.07	6.92	44.34	12.77
1-15-34	354	509	15.0	...	89.0	120.0	29.5	...	2,982	1.11	7.32	46.16	13.61

Table 8.—E197, Data Showing Age, Weight, Feedstuffs Consumed, and Daily Nutrient and Mineral Intake During Experimental Period

Date, end of period	Age	Weight	Feedstuffs consumed							Nutrient intake daily		Mineral intake daily	
			Skim-milk	Prairie hay	Corn	Oats	Corn gluten meal	Dry beet pulp	$CaCO_3$	Crude protein	Total digestible nutrients	Ca	P
	days	lb.	lb.	lb.	lb.	lb.	lb.	lb.	gm.	lb.	lb.	gm.	gm.
6-19-33	137	283	310	58.0	54.0	...	42.0	...	....	1.03	3.39	10.19	8.57
7-19-33	167	322	107	32.0	48.5	42.5	45.5	6.0	....	0.95	4.42	5.58	7.42
8-15-33	194	...	...	...	79.2	18.9	29.7	39.8	....	0.75	4.78	4.75	5.71
8-18-33	197	341	...	...	9.1	2.3	3.4	4.5	195	0.78	4.95	30.86	5.94
9-17-33	227	391	Wheat bran	...	83.5	51.4	38.5	45.0	2,175	0.91	5.52	34.24	7.22
10-13-33	253	399	8.8	...	49.8	42.3	27.0	30.0	1,835	0.77	4.52	32.53	7.90

The rations provided by Rupel, Bohstedt, and Hart (1933) were rather low in phosphorus, which may account for the drop in the inorganic phosphorus of the blood, reported by them. It should also be pointed out that a phosphorus deficiency in the ration may serve as the indirect cause of the disease in cattle under farm conditions. This results from the well-known tendency of cattle on phosphorus-deficient rations to develop a lack of appetite for hay, which, under common winter feeding conditions, is usually the principal source of both calcium and vitamin D.

The Skeleton

The photographs presented constitute abundant evidence of the influence of the syndrome on the skeletons of cattle. The extreme stiffness

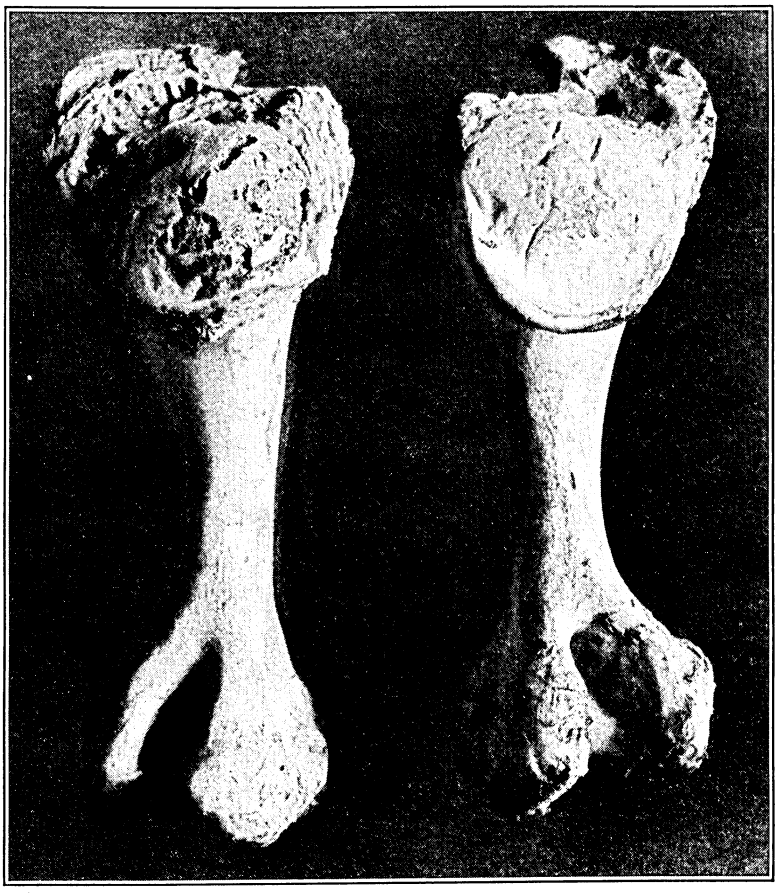

Fig. 7. The Right Humerus from E203 and E187, Respectively, Showing the Effect of the Syndrome on Skeletal Structure

The condition of these bones indicates why complete recovery of affected animals is sometimes impossible.

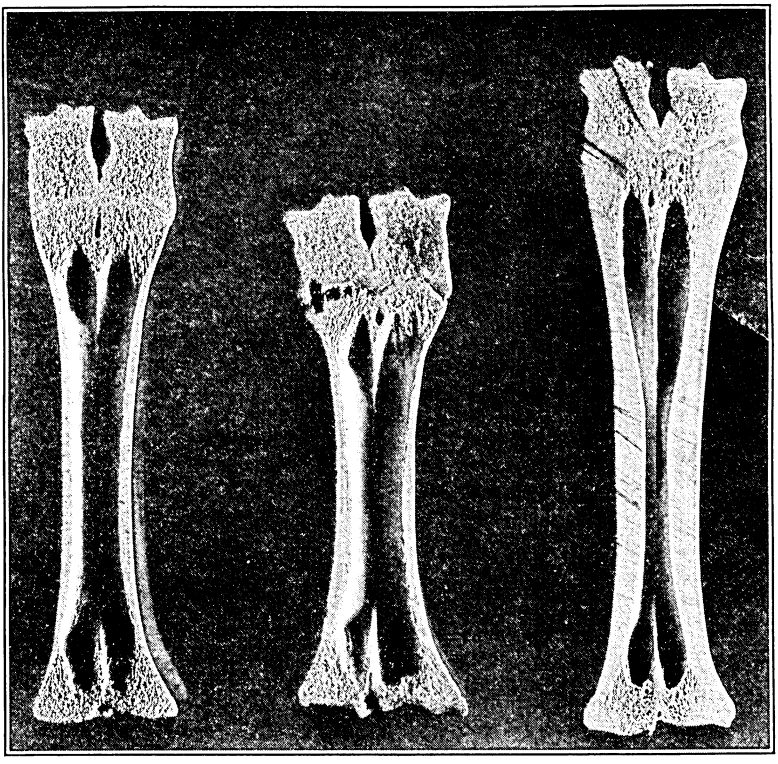

Fig. 8. Effect of the Syndrome on the Structure, Density, and Thickness of Walls of Bones of Cattle

The bone at the right is from a normal animal; the other two were obtained from affected animals.

of the affected animals, the enlarged and swollen joints, the abnormal posture of knees and pasterns, the roughened articulating surfaces of the bones of the joints, as well as the extremely fragile appearance and thin walls of the bones, may be observed.

Figure 7 indicates in a striking manner the effect of the syndrome on the skeletal structure. The larger bone in the photograph was taken from E187. Previous to her slaughter, a grating or crepitating sound as of broken bones rubbing together could be heard when she was made to move about. The condition of this bone explains why she failed to make a complete recovery after receiving an adequate supply of vitamin D and minerals. The other bone was removed from the carcass of E203 at the time of his slaughter. This animal, as is indicated in his life history in the appendix, altho badly affected with the disease in March 1933, at the time of slaughter appeared almost normal. However, it is apparent from the photograph of this bone that complete recovery was

impossible as far as his skeletal structure was concerned. It will be observed that the ball-like end of the bone had become so softened from a lack of proper mineralization that the weight of the animal apparently had caused it to flatten. It is self-evident that such a degree of deformity can never be fully corrected. The serious effects of the disease on the structure and thickness of the bone wall are apparent also in Figure 8.

Chemical analysis was made of the bones of a few of the animals included in the study. Almost all of them had either remained normal during the experiment or had completely or nearly completely recovered from the disease. The bones of only three badly affected animals were subjected to analysis. Of these, E159 showed a serious deficiency of vitamin D, calcium, and phosphorus, and E168, chiefly a deficiency of vitamin D. In both cases the disease was produced experimentally. The third animal was a young steer, brought from a farm in a badly affected condition, probably the result of a deficiency of both calcium and vitamin D. The bones analyzed, in all cases, were the right humerus, femur, and 6th and 11th ribs, except for the young steer from which only the humerus and one rib were taken. The method described by Neal and Palmer (1931) was used for the preparation and analysis of the bones.

Table 9 presents the data obtained from the animals in this study, as well as average analyses from normal and phosphorus-deficient animals obtained in previous experiments (Eckles, Gullickson, and Palmer, 1932).

Considering first the affected animals, it is clear from the data in Table 1 that E159, with an average intake of calcium and phosphorus of only 13.5 grams and 12.5 grams, respectively, per 1,000 pounds of weight and a blood composition of 8.91 milligrams calcium and 2.81 milligrams inorganic phosphorus per 100 cubic centimeters of plasma, was suffering from both calcium and phosphorus deficiency. The higher average calcium and phosphorus intake and blood phosphate analysis of E168 suggests that the mineral deficiency in her ration was not so serious. It seems probable that both animals were suffering from vitamin D deficiency in spite of a moderately liberal intake of hay on the part of E168. No assay was made of the vitamin D activity of the hay. The steer probably represented a typical farm animal fed chiefly on grain during the winter months, resulting in a serious deficiency of vitamin D and calcium. The above suppositions are important for a correct interpretation of the bone data.

The ash content, especially of fresh bones from affected animals, is definitely lowered with increasing complexity of etiology. The ratio of

Table 9.—Bone Composition of Experimental Animals Compared with Normal Controls and Normal and Phosphorus-Deficient Bones

No. of animal	Experimental type	Condition at slaughter	Age at slaughter	Ash content of bones			$Ca_3(PO_4)_2$: $CaCO_3$ ratio
				Fresh	Dry	Dry extracted	
			months	per cent	per cent	per cent	
....	Eight controls from previous experiments	Normal	6 to 24	36.3	48.3	63.0	7.09
E181	Control	Normal	17	35.3	47.9	61.6	6.87
E182	Control	Normal	17	34.2	45.0	62.3	7.91
E180	Control	Normal	18	33.1	44.2	61.6	7.89
E161	Control	Normal	22	33.5	44.0	62.1	6.27
....	Six P deficient animals from previous experiments	Badly affected	11 to 32	27.5	37.5	58.8	5.61
Steer	Naturally occurring case from farm	Badly affected	?	25.5	38.9	59.3	6.75
E159	Experimental, vitamin D, Ca and P deficiency	Badly affected	14	22.7	38.8	55.3	6.06
E168	Experimental, chiefly vitamin D deficiency	Badly affected	15	29.3	42.8	59.8	6.25
E199	Naturally occurring case from farm, $CaCO_3$ 3 months	Nearly normal	10	26.3	50.5	56.1	6.25
E166	Experimental disease, fed $CaCO_3$ 5 months and given sunshine last 3 weeks	Nearly normal	15	29.8	42.8	60.1	6.98
E170	Experimental disease, fed $CaCO_3$ 8 months	Normal	21	32.2	41.0	62.9	6.59
E146	Experimental disease, fed cod liver oil 5 months—sunshine throughout	Nearly normal	25	31.9	41.9	61.2	7.52
E169	Experimental disease, fed viosterol 10 months	Normal	22	32.2	41.1	63.1	7.58
E162	Experimental disease, fed alfalfa hay, viosterol last 2 months	Nearly normal	23	41.6	52.4	64.2	6.56

$Ca_3(PO_4)_2$ to $CaCO_3$ also reflects the introduction of phosphorus deficiency into the eitological picture. Previous experience has shown us that aphosphorosis in cattle invariably causes this ratio to be below 6.00. The bone data are, therefore, in line with the study of mineral intake and blood composition in showing that phosphorus deficiency is not a primary factor either in the naturally occurring syndrome or in that produced experimentally in imitation of the conditions on the farm.

A comparison of the bone data of the animals in which cures had been effected chiefly by calcium administration, with like data from animals that had received direct vitamin administration, seems at first sight to indicate that the latter was more effective in restoring to normal the mineral composition of the bones. However, the comparison is weakened by the fact that the calcium-fed animals received their supplement for a shorter time (on the average) than those fed alfalfa hay, viosterol, or cod liver oil or those that were turned out into the sunshine. One animal in the group fed $CaCO_3$ (E199) had a bone-ash percentage similar to that of the seriously affected animals, in spite of its apparent recovery from stiffness. Obviously, the bone composition is not the cause of the abnormal symptoms, but is one of the effects of the deficiencies. Histological studies have not been made, altho their importance is fully appreciated. The erosion of the articulatory bone surfaces (Figure 7) shows clearly one of the causes of the lameness. It seems probable that future histological studies will show that the disease affecting the bones is either a definite *osteodystrophia fibrosa* or a serious complication with this disorder. According to Theiler (1934), this disease in cattle has not yet been described but is confined to horses, goats, and dogs, and rickets in pigs sometimes is complicated by this osteodystrophy.

PRACTICAL CONSIDERATIONS

Explanation of Occurrence of the Disease

From the facts presented, a simple explanation can be given for the occurrence of the rickets-like disease in calves on farms. Beef-bred calves of the class known as baby beef are most frequently affected. This occurs because of the manner in which they are fed and cared for, not because of a greater susceptibility to the disease than dairy-bred calves. Starting in the fall of the year with a group of feeder calves, recently weaned, the disease probably develops in somewhat the following manner: Such animals, if healthy and thrifty, are growing rapidly with consequent high requirements for minerals and vitamin D. They are fed indoors, where they remain much of the time. They are allowed to consume as much of good-quality concentrates as they desire,

with the result that little hay is eaten, especially since its quality frequently is not of the best. This plan of feeding and care ultimately results in a deficiency of both calcium and vitamin D, as hay is the chief source of both in winter. Even when the animals are exposed to direct sunshine, it probably is of little value in generating vitamin D, in our northern latitudes. This is certainly true in Minnesota.

When the two deficiencies are sufficiently extreme, or if continued for a sufficient period of time, the characteristic symptoms develop. After a time, anorexia develops, which invariably is one of the later symptoms of the disease.

Less concentrates are consumed, with a consequent reduction in the phosphorus intake, which, in turn, results in a phosphorus deficiency as indicated by a fall in the inorganic phosphorus of the blood.

It has been pointed out that the disease occurs more often among feeder calves than in dairy herds. Obviously, this is due to the fact that the dairy herds normally are required to consume considerably more hay than feeder calves and therefore are supplied with a greater amount of vitamin D and calcium. When dairy calves are fed and cared for like beef calf feeders, they also succumb to the disease. This explains why 4-H Club dairy calves are frequently found to be affected.

Prophylactic Measures Required

The disease can be prevented from occurring in young rapidly growing calves only by providing them with an adequate supply of vitamin D and calcium. The form or manner in which it is supplied does not appear to be important; sunshine, viosterol, hay, and cod liver oil have all been found effective sources of vitamin D; feeding good-quality legume hay undoubtedly is the best way to assure an adequate supply of calcium, but some form of calcium supplement will also prove satisfactory for this purpose.

On farms where the disease normally occurs, effort should be made to increase the amount of hay consumed by young cattle, either through improvement in the quality of the product fed or by reducing the amount of concentrates provided. Along with this, effort should be made to expose the animals of the herd to as much direct sunshine as possible. The ability of summer sunshine to produce vitamin D is indicated in the experimental data of E186 during the period from May 21 to October 17 (Table 5). This animal, receiving no hay and a moderately low calcium intake, was protected from the disease by an abundant exposure to sunshine. Likewise, the results from E196 (Table 4) during the period from October 16 to January 15 suggest that even autumn and winter sunshine in east central Minnesota may have some antirachitic

potency for cattle, altho, in all probability, the high calcium intake was an important factor in the recovery of this calf. The wide variation in weather conditions, as well as in the efficacy of the sunshine in different localities during winter months as reported by Clark (1930), Knudson (1932), and Day (1932), suggests that sunshine alone should not be depended on for this protection. Obviously, the amount of hay required to afford protection cannot be given until the information is more complete regarding the relationship of the method of haymaking to the vitamin D content of the product. It is desirable to provide other sources of vitamin D and calcium for cattle on the farm only after those naturally available have been fully utilized and found inadequate.

CONCLUSIONS

From the results of this study, it may be concluded that the characteristic rickets-like disease which occurs in calves during fall, winter, and spring months is caused by failure to provide animals with a sufficient supply of vitamin D and calcium.

Various clinical forms of the syndrome may, and probably do, occur in cattle on farms, depending on the relative degree of deficiency of the two factors.

The disease seriously interferes with normal growth and development. The effect on the skeletal structure is marked, the most striking and most frequent macroscopic features being an enlargement of the joints with considerable erosion of the articulating surfaces and the production of thin fragile walls of the shafts of the long bones.

The onset of the disease is invariably followed by varying degrees of anorexia, a downward trend of the blood calcium and a similar, tho less marked, trend in the inorganic phosphorus of the blood if the environmental conditions remain the same for a sufficient period of time.

The disease may be prevented from developing, or may be corrected if not too far advanced, by providing animals with an adequate supply of calcium and vitamin D. The form or manner in which these products are provided is not important. Sunshine, viosterol, sun-cured hay, and cod liver oil may be used to supply vitamin D; good-quality hay, especially that from legumes, or some supplement rich in calcium may be used to provide the necessary amount of calcium.

LITERATURE CITED

BECHDEL, S. I., ECKLES, C. H., and PALMER, L. S. (1926) The vitamin B requirement of the calf. Jour. Dairy Sci. **9**:409-438.

BECHDEL, S. I., DUTCHER, R. A., and TUCKER, H. H. (1928) The deposition of minerals in the bones of calves fed rachitic and antirachitic rations. Pa. Agr. Expt. Sta. Bull. 230.

BECHDEL, S. I., HONEYWELL, H. E., and DUTCHER, R. A. (1928) The effect of feeding heifers a ration deficient in vitamin A. Pa. Agr. Expt. Sta. Bull. 230, pp. 20-21.

BECHDEL, S. I., HONEYWELL, H. E., DUTCHER, R. A., and KNUTSEN, M. H. (1928) Synthesis of vitamin B in the rumen of the cow. Jour. Biol. Chem. **80**:231-238.

BECHDEL, S. I., and HILL, O. J. (1930) The deposition of minerals in the bones of calves fed rachitic and antirachitic rations. Pa. Agr. Expt. Sta. Bull. 258, p. 25.

BECHDEL, S. I., LANDSBURG, K. G., HILL, O. J., and TUCKER, H. H. (1933) Rickets in calves. Pa. Agr. Expt. Sta. Tech. Bull. 291, pp. 1-41.

BENNETT, FREDERICK. (1932) The deficiencies of milk as an exclusive diet of calves. Thesis, M.S. degree, University of Minnesota.

CANNON, C. Y. (1931) Supplements to a milk diet for dairy calves. Ia. Agr. Expt. Sta. Res. Bull. 136, pp. 65-103.

DAVENPORT, E. (1897) On the importance of the physiological requirements of the animal body: Results of an attempt to grow cattle without coarse feed. Ill. Agr. Sta. Bull. 46, pp. 362-371.

ECKLES, C. H., and SWETT, W. W. (1918) Some factors influencing the rate of growth and the size of dairy heifers at maturity. Mo. Agr. Expt. Sta. Res. Bull. 31.

GULLICKSON, T. W., and ECKLES, C. H. (1927) The relation of sunlight to the growth and development of calves. Jour. Dairy Sci. **10**:87.

HART, E. B., STEENBOCK, H., and ELVEHJEM, C. A. (1924) Dietary factors influencing calcium assimilation. V. The effect of light upon calcium and phosphorus equilibrium in mature lactating animals. Jour. Biol. Chem. **62**:117.

HART, E. B., STEENBOCK, H., ELVEHJEM, C. A., SCOTT, H., and HUMPHREY, G. C. (1926) Dietary factors influencing calcium assimilation. VII. The influence of sunlight upon calcium equilibrium in milking cows. Jour. Biol. Chem. **67**:371.

HART, E. B., STEENBOCK, H., TEUT, E. C., and HUMPHREY, G. C. (1929) Dietary factors influencing calcium assimilation. XI. The influence of cod liver oil upon calcium metabolism of milking cows. Jour. Biol. Chem. **84**:359.

HART, E. B., STEENBOCK, H., KLINE, O. L., and HUMPHREY, G. C. (1930) Dietary factors influencing calcium assimilation. XIII. The influence of irradiated yeast on the calcium and phosphorus metabolism of milking cows. Jour. Biol. Chem. **86**:145.

HUFFMAN, C. F. (1929) Good hay carries factor or factors found in cod liver oil. Mich. Agr. Expt. Sta. Bien. Rept. 1929-30, pp. 18-23.

HUFFMAN, C. F. (1932) A study of the relation of magnesium to calcium, phosphorus and vitamin D in the ration of dairy cattle. Mich. Agr. Expt. Sta. Rept., two years ending June 30, 1932, pp. 23-24.

HUFFMAN, C. F. (1934) Minerals in the nutrition of dairy cattle. Proc. Am. Soc. Animal Prod. pp. 44-49.

INSKO, W. M., and RUPEL, I. W. (1929) Cod liver oil fails to improve a good dairy calf ration. Wis. Agr. Expt. Sta. Bull. 405, p. 50.

JONES, I. R., ECKLES, C. H., and PALMER, L. S. (1926) The rôle of vitamin A in the nutrition of calves. Jour. Dairy Sci. 9:119.

MCCANDLISH, A. C. (1918) Milk as the sole diet of ruminants. Ia. Agr. Expt. Sta. Res. Bull., pp. 3-11.

MCCANDLISH, A. C. (1923) Studies in the growth and nutrition of dairy calves. VI. The addition of hay and grain to a milk ration for calves. Jour. Dairy Sci. 6:347-273.

MORRISON, F. B., and RUPEL, I. W. (1927) Do calves need sunlight? Wis. Agr. Expt. Sta. Bull. 396, p. 39.

MURRAY, A. J. (1912) Diseases of the digestive organs. Diseases of Cattle, Revised edition, U. S. Dept. of Agr., pp. 24-28.

NEAL, W. M., and PALMER, L. S. (1931) A method for the preparation and analysis of representative samples from the bovine skeletal structure. Jour. Agr. Research 42:107-113

OLSON, T. M. (1924) Relative value of whole milk and skimmilk for calves when supplemented with a free choice of six feeds in self-feeders and alfalfa hay ad libitum. Ann. Rept. S. Dak. Agr. Expt. Sta., pp. 17-18.

OLSON, T. M. (1926) Self-feeder for calves. Ann. Rept. S. Dak. Agr. Expt. Sta., p. 15.

OLSON, T M. (1927) Effect of sunlight on growth of calves. Ann. Rept. S. Dak. Agr. Expt. Sta., p. 11.

OLSON, T. M. (1929) Self-feeders in dairy calf feeding. S. Dak. Agr. Expt. Sta. Bull. 236.

REED, O. E., and HUFFMAN, C. F. (1924) Mineral feeding investigations. Mich. Agr. Expt. Sta. Quart. Bull. 7, pp. 14-16.

REED, O. E., and HUFFMAN, C. F. (1926) Feeding of concentrates to dairy cattle. Mich. Agr. Expt. Sta. Quart. Bull. 8, pp. 118-122.

RUPEL, I. W., BOHSTEDT, G., and HART, E. B. (1933) Vitamin D in the nutrition of the dairy calf. Wis. Agr. Expt. Sta. Res. Bull. 115.

SOXHLET, F. (1901) Zeitsch. d. Landw. Prov. Schlesien 44:1575. Cited by Schenke, V. 1903. Phosphorsaurer Kalk als futterbeigabe. Landw. Vers. Stat. 53:291-312.

THEILER, SIR ARNOLD. (1934) The osteodystrophic diseases of domesticated animals. The Vet. Jour. (British) 90:145-175 and 183-206.

THURSTON, L. M., ECKLES, C. H., and PALMER, L. S. (1926) The rôle of the antiscorbutic vitamin in the nutrition of calves. Jour. Dairy Sci. 9:37-49.

THURSTON, L. M., PALMER, L. S., and ECKLES, C. H. (1929) Further studies of the rôle of vitamin C in the nutrition of calves. Jour. Dairy Sci. 12:394-404.

WALLIS, G. C., PALMER, L. S., and GULLICKSON, T. W. (1935) The relation of vitamin D to the retention of calcium and phosphorus in calves as shown by balance trials. Jour. Dairy Sci. 18, pp. 213-228.

ACKNOWLEDGMENTS

The authors wish to acknowledge their obligations to C. H. Eckles, chief of the Division of Dairy Husbandry, for his valuable assistance up to the time of his death, February 13, 1933, in formulating plans for the preliminary study of this problem; to G. C. Wallis, assistant in the Division of Dairy Husbandry, for his assistance in supervising the feeding and care of the animals and for his counsel which aided materially in planning the study; to William G. Werner for his conscientious attention to every detail in the feeding and care of the animals, and to J. W. Nelson and C. E. Rimpila, assistants in the Division of Biochemistry, who carried out the various chemical analyses connected with the investigation, without which a correct interpretation of the experiments would have been impossible.

APPENDIX

LIFE HISTORY OF ANIMALS

E146 Grade Holstein heifer, born 3-22-30. Placed on experiment 11-3-30.

Placed on a low calcium and adequate phosphorus ration beginning 11-3-30. Only 1 lb. of timothy hay was fed daily up to 12-19-30 when 2 lb. were fed to 1-3-31. Approximately 10 gm. $NaH_2PO_4.H_2O$ was provided daily up to and including 12-6-30. Outdoor exercise when weather permitted. Began showing symptoms of stiffness and nervousness during middle and latter part of January 1931. By March was very stiff, being hardly able to move about, lacked appetite, was very nervous, ate soil, and appeared to be blind in one eye. Timothy hay increased again to 2 lb. daily 2-12-31. Began feeding 100 c.c. cod liver oil daily 3-7-31; this was followed by immediate improvement and complete restoration to normal within a few weeks without any other changes being made in the ration. Timothy hay reduced to 1 lb. daily 5-4-31. Cod liver oil supplement removed 8-6-31. Following this she remained normal until late in March 1932 when deficiency symptoms appeared in the form of slight swellings of hocks. During this period of about 7 months her weight increased from about 700 lb. to over 1,000 lb. The swellings on hocks became quite marked by mid-April when the characteristic bent knees also appeared, with more or less lameness. With exposure to more hours of sunshine these symptoms soon disappeared, and at time of slaughter, May 4, 1932, she appeared normal in all outward respects. Weight at time of slaughter, 1,025 lb.

Post mortem revealed slight roughening on articulating surfaces of hock and knee joints with some excess synovial fluid present.

E159 Grade Holstein heifer, born 10-5-30. Placed on experiment 6-1-31.

Ration designed to be low in both calcium and phosphorus. Ration fed during experimental period included from 1 to 3.5 lb. hay (consisting of timothy and prairie in about equal amounts). The larger amount of hay was fed within the period up to 11-14-31; only 1 lb. timothy and 0.5 lb. prairie hay was fed daily from 12-30-31 to end of experiment. Corn, corn gluten meal, starch, and beet pulp made up the remainder of the ration. Outdoor exercise was allowed whenever weather permitted.

On this plan of feeding and management, she continued to thrive until late in September 1931, when signs of unthriftiness began to appear. She ate soil and refused grain occasionally. Her condition very gradually became worse, stiffness developed, legs seemed nearly rigid as she took very short stilted steps when walking, hocks became swollen and knees badly sprung. Appetite was only fair to poor. Molasses was added to ration beginning 12-30-31 in an effort to stimulate appetite. No depraved appetite for old bones. Slaughtered 2-16-32. Post mortem revealed profuse fluid in joints of legs. No enlargement of bones, but quite marked erosion of articulating surfaces of hock and knee joints. Bones rather soft.

E161 Grade Holstein heifer, born 12-23-30. Placed on experiment 7-1-31.

No sunshine after 3-14-32. Fed high calcium ration, slightly low in phosphorus. Original ration included prairie hay, corn, corn gluten meal, beet pulp, starch, and $CaCO_3$. Alfalfa hay fed during period from 1-14-32 to 9-22-32. Molasses and oats also fed during part of experimental period. Remained in excellent physical condition throughout entire life. Slaughtered 10-31-32.

E162 Grade Holstein heifer, born 1-3-31. Placed on experiment 7-1-31.

Fed ration medium to low in calcium, required to low in phosphorus. Original ration included prairie hay, corn, corn gluten meal, beet pulp, and starch; some molasses, alfalfa hay, and oats were also fed. Twelve gm. $CaCO_3$ was provided daily from 10-16-31 to 11-14-31 and 8 gm. daily from 2-12-32 to 3-9-32. Developed various deficiency symptoms as early as 12-15-31 and was quite stiff by 3-12-32. Alfalfa hay feeding began 1-14-32, but considerable prairie hay was also fed to 3-14-32. Kept out of sunshine after 3-14-32. Increased amount of alfalfa hay fed 3-15-32. Improving 3-28-32. Physical condition bad 4-13-32; stood humped, knee-sprung, joints swollen. Alfalfa hay increased to about 12 lb. daily. Improving 4-30-32. Improving very slowly, still showed symptoms 6-8-32. Good condition of flesh, but still sprawling action 7-25 to 9-1-32. Turned out-of-doors in sunshine daily 9-1-32. Viosterol feeding (5 c.c. daily) began 10-6-32. Experiment discontinued 12-21-32. Never completely restored to normal appearance due to permanent changes that had apparently taken place.

E164 Grade Holstein heifer, born 1-21-31. Placed on experiment 8-30-31.

Ration very low in calcium, roughage limited to 1 lb. timothy hay. First deficiency symptoms—nervous, excitable, late October. Humped back, rapid respiration 11-25-31. Timothy hay increased to 2 lb. daily 11-28-31. Kept out of sunshine beginning 12-5-31. From this time on showed gradual decline, stiffness, paddling action of front legs, swollen hocks, waning appetite, and unthrifty appearance. This decline was aggravated when hay fed was again reduced to 1 lb. on 3-10-32. $CaCO_3$ added 3-23-32 with immediate improvement. Normal except for almost constant bloating 9-1-32. Experiment discontinued 9-1-32.

E166 Grade Holstein heifer, born 2-6-31. Placed on experiment 8-30-31.

Same plan of feeding as for E164; i.e., very low calcium and roughage limited to 1 lb. timothy hay daily, with adequate phosphorus. Remained normal until about middle of November 1931. Showed nervousness, starry eyes, wild, labored respiration, more or less stiff, with paddling gait, back humped and middle tucked up. Lacked complete control over rear legs. Blood composition still normal. Twenty-five gm. $CaCO_3$ added daily beginning 11-24-31. Improvement followed, but blood calcium began to decline. Kept out of sunshine beginning 12-5-31. No marked change to 1-1-32, then previous deficiency symptoms began to reappear. Blood Ca down to 6.03 mgm. per 100 c.c. plasma. $CaCO_3$ supplement gradually increased to 100 gm. daily January 8 to 12, inclusive. Immediate improvement. By middle of March seemed normal except for lack of complete control of rear limbs. Exposed to sunshine daily beginning 4-15-32. Did not recover complete control of rear limbs. Slaughtered 5-4-32. Post mortem revealed no marked abnormalities. Spinal processes soft. No erosion in joints.

E168 Grade Holstein heifer, born 2-22-31. Placed on experiment 10-16-31.

Same plan of feeding as E162; i.e., moderate calcium with 2-4 lb. prairie hay daily, in addition 1 to 1½ lb. alfalfa hay was also fed from 1-14-32 to 2-9-32, inclusive. Remained normal to about 2-15-32, then showed stiffness, lack of appetite, stilted action, straight hocks. Condition severe by 3-15-32, and amount of Ca fed increased to 50 gm. per 100 lb. weight by adding about 40 gm. $CaCO_3$; also increased amount prairie hay fed. Showed slight temporary improvement. Kept out of sunshine after 3-15-32. Grew worse again, went down and nearly collapsed on 5-9-32, then tried viosterol feeding, with some improvement in ambition. Dur-

ing vigorous efforts to get up, bones of hocks became exposed. Slaughtered 5-13-32. Vital organs and viscera normal. Some slight erosion on surfaces of hock and knee joints.

E169 Grade Holstein heifer, born 3-30-31. Placed on experiment 11-28-31.

Ration—low calcium, required phosphorus. Original ration included approximately 2 lb. hay (timothy and prairie) daily, with corn, corn gluten meal, beet pulp, and starch sufficient to provide the nutrients required. Timothy hay was discontinued 1-13-32. Prairie hay increased to 3.5 lb. daily 1-14-32, but reduced to 2.5 lb. 2-12-32.

Remained outwardly normal up to 3-15-32, then showed slight stiffness. Kept out of direct sunshine beginning 3-16-32. Stiff and humped 3-21-32. Prairie hay increased to 4 lb. daily. Decreased appetite; small amount of molasses added. Very stiff and decrepit, hardly able to get up 3-26-32. Viosterol (Abbot. Lab. 50D) feeding begun 3-27-32 at rate of 5 c.c. daily. Showed decided improvement physically, appetite better 4-1-32. Only slightly stiff 4-7-32. Appeared entirely normal physically 4-13-32. Viosterol reduced to 2 c.c. 5-16-32. Prairie hay gradually increased to 6 lb. Continued entirely normal until discontinued 1-20-33.

E170 Grade Holstein heifer, born 4-17-31. Placed on experiment 12-28-31.

Plan of feeding similar to that of E169, with approximately 2.5 lb. hay (prairie and timothy) fed daily. Timothy hay discontinued 1-13-32 and prairie hay increased to 3.5 lb. daily 1-14-32, but reduced to 2.5 lb. 2-12-32. No sunshine after 3-26-32. Prairie hay increased to 4 lb. daily 3-23-32. Outwardly normal up to about 4-12-32, when she began to show slight stiffness. Quite stiff by 4-19-32. Began feeding 100 gm. $CaCO_3$ daily 4-28-32. Improvement followed and stiffness completely disappeared by 5-9-32. From this time until discontinued on experiment 1-20-33 she had short intermittent periods of stiffness, some of which probably were due to injuries from rough contacts with other animals during exercise.

E180 Grade Holstein heifer, born 12-18-31. Placed on experiment 8-24-32.

Ration included from 3 to 6 lb. of prairie hay and provided approximately 30 gm. Ca, supplied in part by $CaCO_3$ from 11-22-32 and on, and 15 gm. P per 1,000 lb. of weight, with no exposure to sunshine. The phosphorus was supplied from 4-21-32 and on, in part by $NaH_2PO_4.H_2O$. On this plan of feeding and management she continued normal in all respects until slaughtered 6-23-33. Post-mortem examination revealed a normal condition in all respects.

E181 Grade Jersey heifer, born 12-20-31. Placed on experiment 8-24-32.

Same plan of feeding as for E180 but with daily exposure to sunshine. On this plan of feeding and management she remained normal in all outward respects. She was slaughtered 5-8-33. Autopsy showed normal condition.

E182 Grade Holstein heifer, born 1-24-32. Placed on experiment 8-24-32.

On same plan of feeding as E180, but with 2 c.c. viosterol added daily. On this plan of feeding and management she remained normal in all outward respects. She was slaughtered 6-23-33 in excellent condition.

E185 Grade Guernsey heifer, born 5-29-32. Placed on experiment 11-22-32.

On ration providing approximately 30 gm. each of Ca and P per 1,000 lb. of weight. No direct sunshine, but 3 to 4 lb. prairie hay daily, with necessary

amounts of corn, corn gluten meal, and occasionally beet pulp, wheat bran, and oats. On this plan of feeding and care, she remained normal until 5-20-33. She was then started on a ration that was gradually made hayless, with beet pulp provided for roughage. No sunshine. Mineral intake was that normally provided by this type of ration.

Hay reduced to 1 lb. (prairie) daily 6-7-33. Stiff, knees bent forward, back humped 6-22-33. Hay feeding discontinued entirely 7-6-33. Poor appetite, pica, stiff 7-12-33. Very stiff, humped, knees bent, poor appetite, 8-8-33. $CaCO_3$ supplement added 8-10-33. Slightly improved and better appetite 8-17-33. Growing much worse 9-6-33. Very bad condition 9-21-33. Viosterol added (5 c.c. daily) 9-26-33. Showed marked improvement in appearance and appetite 10-5-33. Discontinued 10-17-32; was still slightly stiff but improving daily.

E186 Grade Guernsey heifer, born 6-15-32. Placed on experiment 12-22-32.

On same plan of feeding as E185, but allowed exposure to direct sunshine. Like E185, she remained normal on this plan of feeding and care. Beginning 5-21-33, she was gradually changed to a hayless ration similar to that of E185. She was exposed to 4 to 8 hours of direct summer sunshine daily. Hay was reduced to 1 lb. (prairie) on June 8 and discontinued entirely 7-6-33. She remained normal in all outward respects during the entire period while on experiment. Experiment discontinued 10-17-33.

E187 Grade Holstein heifer, born 6-28-32. Placed on experiment 1-21-33.

Ration provided approximately 50 gm. calcium daily per 100 lb. of liveweight, with required phosphorus. No sunshine. Ration consisted of approximately 5 lb. prairie hay, with corn, corn gluten meal, beet pulp, and $CaCO_3$ supplement. On this plan of feeding and management she remained normal in all outward respects.

On June 21 a new plan of feeding was begun. All hay was discontinued from the ration, and beet pulp was increased to as much as she would eat. The level of phosphorus intake was also somewhat reduced.

Within a month pica developed, as indicated by a desire to chew boards of pen. Her appetite was only fair. Stiffness developed gradually. Showed first evidence of cocked ankles about 9-6-32; by 10-16-32 stiffness was evident and by 10-29-32 she was quite stiff and decrepit.

Forty gm. $NaH_2PO_4.H_2O$ added to ration daily beginning 11-4-32. This was continued up to and including 11-22-32. At the end of that time she was in very bad physical condition, very stiff and decrepit, middle drawn up, right shoulder badly swollen, got up with great difficulty, knees were badly bent, and a grating sound could be heard as she moved about. Her appetite was very poor. Decided something had to be done at once to prevent her death. 11-22-33 began feeding alfalfa hay about 4 lb. daily, also injected 5 c.c. viosterol daily (11-21 to 11-26 incl.) via jugular vein; later this amount of viosterol was given in the feed. From 11-26-32 to 12-2-32, inclusive, 100 gm. $CaCO_3$ was fed daily, otherwise only 55. By 12-16-32 she showed some improvement, but stiffness still remained and grating sound could be heard in joints of shoulder and hip as she moved about. The viosterol was reduced to 3 c.c. daily 12-17-32. The stiffness and grating of joints continued but her appetite improved 2-5-34. Viosterol feeding was discontinued 2-8-34, and she was placed on normal type of ration including alfalfa hay fed ad libitum, 2 lb. corn and 2 lb. oats. She remained stiff and decrepit, with creaking joints. Slaughtered 3-5-34.

Post-mortem examination revealed joints to be in a very serious condition, especially those of shoulders and hips. Articulating surfaces were very deeply eroded (see Fig. 7), which no doubt accounted for the grating sound heard as she attempted to move about. None of the bones had been broken, but the walls of many of them were very thin. Ends of bones in general were enlarged and appeared as tho they had been too soft to withstand the pressure from the weight of the body. In other respects the carcass appeared normal.

E190 Grade Holstein heifer, born 7-30-32. Placed on experiment 1-21-33.

On ration providing approximately 100 gm. Ca daily per 1,000 lb. liveweight, with phosphorus intake slightly below requirement. No sunshine. Ration provided approximately 5 lb. prairie hay, with corn, corn gluten meal, beet pulp, starch, and the needed amounts of $CaCO_3$. Remained normal in all outward respects. This plan of feeding was discontinued 6-21-33. The prairie hay was discontinued and the mineral intake was controlled at approximately 50 gm. Ca and 12 gm. P per 1,000 lb. of weight. No sunshine. Under this treatment she gradually became stiff and decrepit and lost her appetite. Blood plasma analysis suggested more of a phosphorus deficiency than of calcium.

Beginning 10-8-33 she was turned out of doors several hours daily to prevent her complete collapse. Considerable improvement followed even tho the amount of effective sunshine was very limited. The blood calcium, however, remained the same (slightly low), with the inorganic phosphorus declining to 2.49 mgm. per cent. During the period from 12-17-33 to 1-9-33 a total of 98 lb. of alfalfa hay was fed. Discontinued on experiment 1-9-34. Blood plasma analysis made 1/9-11 indicated that mineral content had been restored to normal, 10.88 mgm. per cent Ca and 6.02 mgm. per cent inorganic phosphorus. In outward respects also she was nearly normal and improving daily.

E194 Holstein freemartin, born 7-13-32. Placed on experiment 1-21-33.

No sunshine. Ration at start provided 5 lb. prairie hay, with necessary amounts of corn, corn gluten meal, beet pulp, and starch. This ration supplied about 31 gm. Ca and 18 gm. P daily per 1,000 lb. of weight up to 3-21-33. From 3-22-33 to 5-20-33 only 4 lb. prairie hay was consumed daily, with the calcium and phosphorus intakes dropping to 18.5 and 16.0 gm. daily, respectively, per 1,000 lb. liveweight. On this plan of feeding and care, she remained normal. Plan of feeding was changed gradually, beginning 5-21-33, to a hayless ration similar to that of E185, E186, and E190. Only 2 lb. of prairie hay was fed daily from 5-21-33 to 7-6-33 and it was discontinued entirely 7-19-33. On this plan she showed slight stiffness by 7-5-33, with appetite waning by 7-31-33. She was very stiff by 8-8-33, being hardly able to get about, with humped back, bent knees, and poor appetite. Beginning 8-10-33, 100 gm. $CaCO_3$ daily was added to ration. This resulted in brief periods of slight improvement, especially in appetite, but with constant bloating. Following this, her condition gradually became worse, hocks became swollen, and other symptoms appeared in aggravated form. On 9-26-33 5 c.c. viosterol was added daily to ration, with the 100 gm. $CaCO_3$ which was being continued. This was followed by immediate improvement in appetite and physical well-being. Viosterol supplement discontinued 11-2-33 and placed on normal ration. Slaughtered 12-1-33. Practically normal in action and appearance. Bones nearly normal in appearance, macroscopically.

E196 Holstein freemartin, born 1-26-33. Placed on experiment 5-21-33.

On ration designed to be hayless and to provide a mineral intake similar to that provided calves that become affected with syndrome on farms. No direct sunshine. Prairie hay was fed at the rate of approximately 1 lb. daily up to 7-19-33, after which it was discontinued entirely. Other feedstuffs included in ration were, skimmilk up to 7-3-33, with the necessary amounts of corn, corn gluten meal, oats, and beet pulp which replaced the hay. Showed stiffness by 7-19-33 or before all the hay was discontinued from ration. Bloated, showed poor appetite, was stiff, humped up, and nervous, and had several fits, 7-31-33 to 8-7-33. Began feeding 75 gm. $CaCO_3$ daily 8-8-33. This was followed by improvement for a time, but by 10-15-33 was in precarious condition, near collapse. A total of 8 lb. prairie hay was fed 10-14-33 to 10-17-33, inclusive. She was turned out-of-doors daily beginning 10-16-33. Improvement followed immediately, and this continued until removed from experiment on 1-15-34 in what appeared to be normal physical condition.

E197 Grade Holstein heifer, born 2-2-33. Placed on experiment 5-21-33.

Conditions identical with those of E196, and identical results. The $CaCO_3$ supplement was added to the ration 8-16-33, with results similar to those obtained with E196. She died during an attack of fits 10-14-33. Autopsy showed bones thin-walled, but joints near normal.

E198 Grade Holstein heifer, born 2-12-33. Placed on experiment 5-21-33.

Ration included prairie hay fed ad libitum and provided approximately the same amount of minerals as the rations of E196 and E197. No sunshine. On this plan of feeding and care she remained normal in all outward respects during the entire time on experiment. Discontinued 12-16-33.

E199 Grade Hereford heifer, about six months old when obtained from farmer, February, 1933.

Badly affected with rickets-like syndrome at time of arrival. (See Fig. 1.) Could not get up alone. Fed ration similar to the one she had received on farm, 1 to 1.5 lb. prairie hay with 100 gm. $CaCO_3$ added daily. No sunshine. Showed very gradual but continuous improvement on this plan of feeding. Experiment discontinued 5-20-33.

E202 Grade Shorthorn bull, about seven months old when obtained 3-2-33 from farmer near Chokio, Minnesota.

Was able to move about, but quite stiff, with bent knees, and lacked appetite. First few days was placed on ration similar to that received on farm. This included about 1.5 lb. prairie hay, with corn, corn gluten meal, and oats. No sunshine. Showed little or no change in appearance and physical condition. Beginning 3-10-33, added 35 gm. daily of $NaH_2PO_4.H_2O$ to determine to what extent P is factor, also began feeding approximately 1.25 lb. wheat bran on same date. Condition immediately became worse; went down, unable to get up alone. Discontinued feeding P supplement, also the wheat bran, 3-19-33. Began feeding $CaCO_3$ at rate of 100 gm. daily 3-20-33. Improvement followed immediately but gradually. He was quite normal when slaughtered 10-4-33, altho post mortem revealed unusually soft bones. Ribs enlarged ventrally. Ends of humeri and femurs greatly enlarged, with articulating surfaces extremely eroded and misshapen as tho pressed out from weight carried.

E203 Grade Shorthorn bull, about eight months old.

Statements made about E202 also apply to this animal. Same plan of feeding and care followed as with E202. Feeding of about 40 gm. $NaH_2PO_4.H_2O$ was begun 3-15-33, with results similar to those obtained with E202. No wheat bran was fed. Phosphorus supplement discontinued and calcium supplement in form of 100 gm. $CaCO_3$ added, beginning 3-20-33, with results similar to those of E202. Slaughtered 10-4-33. Condition same as that of E202.

E204 Grade Shorthorn bull, about seven months old. Obtained same time and place as E202 and E203.

Was in very bad condition at time of arrival. Placed on ration similar to that fed on farm, including from 4 to 8 lb. skimmilk, 2 lb. prairie hay, corn, oats, corn gluten meal, and wheat bran. Kept indoors, out of direct sunshine. He grew worse; was soon unable to get up and could not stand alone. Lacked ability to adjust or move rear legs; hock joints would interlock. Helped to get on feet several times daily. Respiration very rapid. At this stage (3-10-33) the feeding of 100 gm. $CaCO_3$ daily was begun. Improvement was noted immediately, and within a week he was able to get up and stand alone and a few days later could move about a little. Improvement continued until he was nearly normal. (See Fig. 3.) Slaughtered 10-4-33. Post-mortem examination revealed condition similar to that of E202.

E206 Grade Guernsey bull, about six months old when obtained from farmer near Anoka, Minnesota.

This animal had previously been very stiff and had shown other rickets-like symptoms, but was somewhat improved at time he was purchased He was placed on a ration similar to that received when he became affected. This included about 1 lb. prairie hay daily, with the necessary amount of a grain mixture made up of 2 parts oats and 1 part each of corn and corn gluten meal. Four lb. of skimmilk was also provided daily during the first 8 days. No sunshine. Under these conditions he became buck-kneed and developed other characteristic symptoms, bloated constantly, and had to be tapped 4-30-33; also had several attacks of fits. (See Fig. 2.) Prairie hay increased to about 1.5 lb. daily 5-11-33. Began feeding 25 gm. $CaCO_3$ 6-8-33 as he was very stiff and decrepit. This was gradually increased to 100 gm. by 6-22-33. Improvement followed immediately and continued. He appeared entirely normal when sold for slaughter 10-17-33. During the improvement, the hay consumption increased gradually to about 2 lb. daily. This may have been a slight factor in his improvement. The blood calcium rose to its highest point (10.76) on July 13-16, 1933. Following this, it declined again. At the time of disposal the calcium and inorganic phosphorus of the blood plasma were 8.71 and 7.09 mgm., respectively, per 100 c.c. The hay consumption had now risen to about 3 lb. daily.